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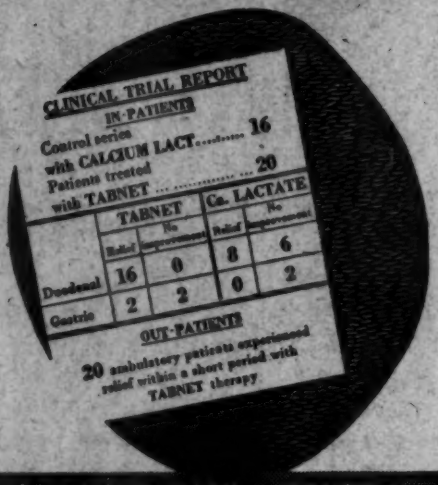

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BRITISH MEDICAL JOURNAL

LONDON SATURDAY OCTOBER 17 1953

CHRONIC GASTRIC ULCER

A COMPARISON BETWEEN A GASTROSCOPICALLY CONTROLLED SERIES TREATED MEDICALLY AND A SERIES TREATED BY SURGERY

BY

B. F. SWYNNERTON, D.M., M.R.C.P.

Weir Research Assistant, Wandsworth Hospital Group

AND

N. C. TANNER, F.R.C.S.

Senior Surgeon, St. James's Hospital, London

Chronic duodenal and gastric ulcers are usually studied together under the title of "peptic ulcer," but there is evidence to suggest that their aetiology is different. Duodenal ulcer is predominantly a disease of young adults with a thick hypersecreting gastric mucosa, while gastric ulcer is commonest in the middle-aged with a normal or thinning mucosa and is associated with normal or diminished acidity. The treatment of duodenal ulcer is to some extent physiological in that, whether by neutralization, vagotomy, or partial gastrectomy, the effect is to reduce the high acidity. In the case of gastric ulcer, however, the application of the same principles is less rational, although, at present, the only effective form of treatment. These considerations make it advisable to study them separately.

For economic and clinical reasons there has been an increasing tendency towards the use of surgery in the treatment of chronic gastric ulcer. It is no longer possible in larger centres to compare the late results in controlled series, the one treated medically and the other surgically, but some useful information may be forthcoming from an analysis of uncontrolled material.

Present Investigation

The present investigation concerns 498 patients with chronic gastric ulcer. They are divided into two series—one treated medically and the other surgically in the gastro-enterological clinic at St. James's Hospital during the years 1940–6 inclusive. Late information concerning nearly all was obtained in 1952, giving a minimum follow-up period of five and a maximum of twelve years.

There are 262 patients in the medical series, in which the criterion for inclusion was the presence of a gastroscopically confirmed *active* chronic gastric ulcer. In view of the rarity of the combination of even quiescent duodenal ulcer with gastric *carcinoma* (only two have been encountered in over 2,200 gastrectomies in ten years at St. James's Hospital) and the need for more evidence on the "ulcer-cancer" problem, those patients in whom a previous or concomitant duodenal ulcer was suspected have been omitted. Also excluded are those patients who were submitted to a curative operation within one year of gastroscopic diagnosis. Analysis of this series may be expected to reveal a preponderance of patients with short histories or mild symptoms, of those who respond rapidly to treatment and relapse rarely, of those who by reason of age or poor general condition are considered to be unsuitable for surgery, and of those who refuse operation.

In the surgical series there are 254 cases. Patients with additional duodenal ulcers or scars are not included. Those in whom the only operation was repair of a perforated ulcer are excluded, since this cannot be regarded as a curative procedure and since the localization of juxtapyloric perforations may be difficult at emergency operation. Because this is a clinic specializing in gastric disorders the surgical series contains a proportion of technically difficult cases which had been referred from elsewhere. For example, the incidence of high lesser-curve ulcers will be greater than is normally found.

Eighteen patients, sixteen men and two women, appear in both series.

Age and Sex

Of the 498 patients 387 were men and 111 were women, a ratio of 3.5:1, which is the usual recent finding, but a reversal of earlier statistical figures. The reason for this change is the great reduction in the incidence of gastric ulcer in *young* women.

It is not possible to give complete information on the ages at which the diagnosis of gastric ulcer was first made, since in a certain number of cases this was done by *x*-ray examination of a barium meal previous to the time of gastroscopy. Table I does, however, give some indication, and shows the age distribution of cases at

TABLE I.—*Gastric Ulcer: Age and Sex Incidence*

Age (years)	Medically Treated				Surgically Treated			
	Men		Women		Men		Women	
	No.	%	No.	%	No.	%	No.	%
10–19	—	—	1	1·4	—	—	—	—
20–29	7	3·7	1	1·4	2	0·9	—	—
30–39	17	8·9	6	8·5	27	12·7	2	4·8
40–49	51	26·7	15	21·1	56	26·4	9	21·4
50–59	51	26·7	26	36·6	86	40·6	21	50·0
60–69	53	27·7	17	23·9	37	17·5	7	16·7
70–79	12	6·3	5	7·0	4	1·9	3	7·1
Total	191		71		212		42	

Note: Combined figures are not given, since 16 men and 2 women appear in both series.

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the start of the investigation, thus serving as a useful baseline. In addition it shows the ages at which patients came to operation.

It is unusual for patients with chronic gastric ulcer to present at hospital before the age of 30, but after this there is a steady increase in numbers until the middle fifties. As might be expected, most of the youngest and oldest patients were treated conservatively. In both sexes the highest operation rate was in the sixth decade and a higher proportion of men than of women were treated by surgery.

Age at Onset of Symptoms and Length of History

The average age at the onset of symptoms was 43.6 years in men and 44.6 years in women. The similarity between the two sexes is clearly shown in Table II, where the series are amalgamated. These figures confirm the fact that, although gastric ulcers may start at any age, they most commonly do so after the age of 30.

In Table III a comparison is given of the length of symptoms in the two series. It must again be borne in mind that in the medically treated series the times given are not necessarily those of the initial diagnosis. Amongst the men one finds that those with short histories tend to be treated medically. Why this does not hold good for women

TABLE II.—*Gastric Ulcer: Age at Onset of Symptoms*

Age at Onset (years)	Medical and Surgical				Men and Women	
	Men		Women			
	No.	%	No.	%	No.	%
10–	9	2·3	7	6·3	16	3·2
20–	48	12·4	7	6·3	55	11·0
30–	79	20·4	19	17·1	98	19·7
40–	115	29·7	33	29·7	148	29·7
50–	75	19·4	28	25·2	103	20·7
60–	35	9·0	11	9·9	46	9·2
70+	8	2·1	2	1·8	10	2·0
Unstated	18	4·7	4	3·6	22	4·4
Total	387		111		498	

TABLE III.—*Gastric Ulcer: Length of History*

Length of History (years)	Medically Treated				Surgically Treated			
	Men		Women		Men		Women	
	No.	%	No.	%	No.	%	No.	%
Under 1	40	20·9	7	9·9	21	9·9	7	16·7
1–3	25	13·1	14	19·7	20	9·4	10	23·8
3–5	26	13·6	8	11·3	28	13·2	7	16·7
5–10	32	16·8	13	18·3	51	24·1	8	19·0
10–20	31	16·2	12	16·9	52	24·5	4	9·5
Over 20	28	14·7	15	21·1	30	14·2	4	9·5
Unstated	9	4·7	2	2·8	10	4·7	2	4·8
Total	191		71		212		42	

Note: The majority of the "unstated" cases fell into the longer history groups. They were recorded as "many years."

is not clear. It would seem that women, if they do not have an operation early in their ulcer history, become adapted to its presence better than men, possibly because they can, for domestic reasons, treat their recurrences more thoroughly. There is a falling off of the operation rates in men with a history of over 20 years. These too will have learnt to "live with their ulcer," and for this reason their appearance at hospital should excite suspicion that recent symptoms which they attribute to their ulcer may be due to other causes (Tanner, 1951).

Sites of Chronic Gastric Ulcer

The impression in this clinic that the high posterior aspect of the lesser curve is the site of predilection for chronic gastric ulcers in women is confirmed (Table IV). There is a pronounced drop in the frequency as the lower stomach is approached, and ulcers in the pyloric antrum and canal are exceptional. This last observation has been made on several previous occasions (see Ivy, Grossman, and Bachrach, 1951), In men, on the other hand, the distribution of ulcers from the cardia to the pylorus is more even, with the highest incidence in the mid and lower body, and over 10% are found distal to the angulus. The reasons for these differences between men and women are unknown.

TABLE IV.—*Sites of Chronic Gastric Ulcer*

Vertical					Transverse				
Site	Medical and Surgical				Site	Medical and Surgical			
	Men		Women			Men		Women	
	No.	%	No.	%		No.	%	No.	%
High body ..	78	19·2	52	43·3	Posterior lesser curve	119	29·3	53	44·2
Mid body ..	109	26·8	39	32·5	Lesser curve	122	30·0	47	39·2
Low body ..	88	21·7	20	16·7	Anterior lesser curve	33	8·1	10	8·3
Angulus ..	74	18·2	7	5·8	Greater curve ..	1	0·2	1	0·8
Pylorus and antrum ..	55	13·5	1	0·8	Angulus ..	74	18·2	7	5·8
Unknown ..	2	0·5	1	0·8	Pylorus and antrum ..	55	13·5	1	0·8
Total	406		120		Unknown	2	0·5	1	0·8
					Total	406		120	

Note: 2 men had three ulcers each. 15 men and 9 women had two ulcers each.

TABLE V

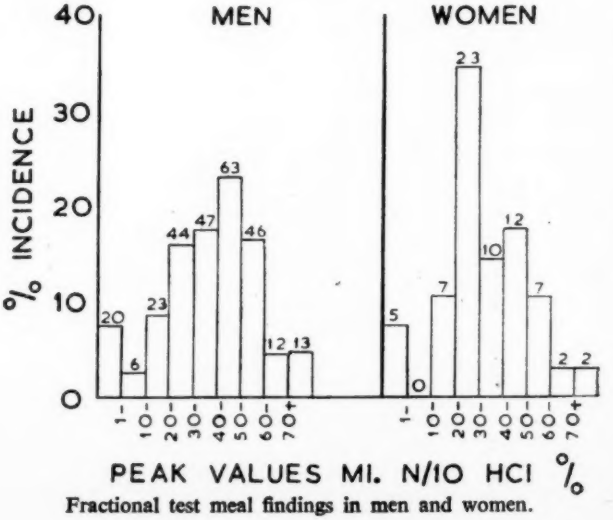
	High Lesser Curve			Mid Lesser Curve			Low Lesser Curve		
	Ant.	Mid.	Post.	Ant.	Mid.	Post.	Ant.	Mid.	Post.
Men ..	3	31	44	6	53	49	24	38	26
Women	3	20	29	6	16	17	1	11	7

In the body of the stomach the anterior lesser curve and anterior wall of the stomach are comparatively immune to peptic ulceration, while the posterior lesser curve is very susceptible (see Table V). In men the differences in anterior and posterior ulceration decrease as the angulus is approached, whilst in women the high proportion of posterior ulcers is maintained.

Fractional Test Meal

Results of a fractional test meal, using gruel as the stimulant, were available in 342 patients. The peak values of free hydrochloric acid measured as millilitres of N/10 HCl are shown in the Chart.

The highest incidence in men was with free acidity between 40 and 50 ml. (63), and in women 20 to 30 ml. (23). The age of the patient at the time of the test meal makes little difference to the findings, and it is only by gross



Fractional test meal findings in men and women.

grouping that any higher incidence of hypochlorhydria in the older patients can be shown (Table VI).

Amongst the women, in whom the numbers are smaller, the age of the patient seems to be completely unrelated to the test-meal findings. It seems to be the presence of a degenerate mucosa rather than the age of the patient which leads to the formation of a gastric ulcer, though age indirectly affects the incidence in so far as mucosal atrophy increases with increasing years.

TABLE VI.—*Chronic Gastric Ulcer: Acidity in Relation to Age (Men Only)*

Peak Values ml. N/10 HCl	Men under 50		Men over 50	
	No.	%	No.	%
0–19	15	14·3	34	20·1
20–39	35	33·3	56	33·1
40–59	41	39·0	68	40·2
Over 60	14	13·3	11	6·5
Total	105		169	

Although there are differences in the fractional test meal findings when they are related to the site of the ulcer in the stomach, they are not great, and it has not proved possible to work out any definite correlation.

Comparison between different series of gastric analyses is well-nigh impossible owing to different techniques used and differing interpretations put upon the results. The results in this investigation are given here, since the gruel test meal is probably the type of gastric analysis most widely used in this country.

Early Results of Treatment

Medical Series

The unceasing flow of papers to the journals shows that there is no unanimity concerning the effectiveness of individual methods of treating gastric ulcers. Suffice it to say that nearly all will heal on rest in bed, regular feeds, and antacids, which were the general principles adopted in this series. Five men and one woman died during their initial stay in hospital—three of haemorrhage from their ulcers, two of pneumonia, and one of bronchial carcinoma.

The normal procedure is for a patient suspected of having a gastric ulcer to be gastroscoped within a few days of first being seen and then at regular intervals—usually fortnightly. Patients are kept under a strict regime until there is evidence, preferably gastroscopic, that the ulcer is healed. The Schindler gastroscope was used for the earlier cases and the Hermon Taylor gastroscope for the later ones.

Surgical Series

The procedures and immediate results in this series, which includes emergency operations, are shown in Table VII.

TABLE VII.—*Surgical Series: Procedure*

Procedure	No.	Deaths	Mortality
Local excision	4	3	75·0%
Gastro-jejunostomy	12	3	25·0%
Partial gastrectomy (whole series)	238	15	6·3%
Pastial gastrectomy (excluding 1940–1)	175	5	2·9%

Note: Operative deaths are taken as all deaths occurring within one month of operation.

In 1940 and 1941 there were 13 operative deaths (10 following partial gastrectomy, 3 following gastro-jejunostomy), and the figures for these two years account for the high mortalities. All these deaths occurred during the first five months of intensive night bombing on London, and, although the causes of death seemed to bear no direct relationship to the bombing, there can be little doubt that they were due to resistance being lowered as a result of stress (Tanner, 1946, 1951).

Many of these patients were operated on in the pre-penicillin era, and it is of interest to compare the figures with the more recent results. In the last 350 partial gastrectomies performed by one of us (N.C.T.) before 1953 for chronic gastric ulcer, unaccompanied by duodenal ulcers or scars or by oesophageal ulcers, there have been four operative deaths—an incidence of just over 1%. These include emergency operations for haematemesis, of which there were 38 cases with two deaths.

Surgery in the treatment of gastric ulcer may be expected to carry a higher mortality than for duodenal ulcer, since one is dealing with an older age group. Table VIII shows the correlation between the age of the patient and the operative mortality.

TABLE VIII.—*Surgical Series: Age of Patients Dying Post-operatively*

Age (years)	No. in Series	Died	
		No.	%
40–	65	2	3·1
50–	107	9	8·4
60–	44	7	15·9
70–79	7	3	42·9

Unless there is some strong indication for surgery, patients over the age of 65 are treated medically in this clinic, and the older age groups contain a high percentage treated by emergency operation for uncontrolled haemorrhage.

Local excision of ulcers was practised only as an emergency operation for those patients in dire straits from uncontrolled haemorrhage. Gastro-jejunostomy was reserved for the feeble and elderly with obstructive symptoms. Partial gastrectomy was the operation of choice both as an emergency and as a planned procedure. The type most frequently performed was the antecolic Polya–Hofmeister (196 cases), with or without a Pauchet extension, depending upon the site of the ulcer. In 1946 the Billroth I type of partial gastrectomy was used more often, and has since become the operation of choice for chronic gastric ulcer, as it is a simpler and perhaps more physiological procedure.

Method of Follow-up

A circular letter containing a questionary was sent to the last known address of the patient requesting him to attend at the clinic if possible, or, if unable to do so, to complete the questionary. In the event of failure to receive a reply to this and a postcard reminder, or the letter being returned as "unknown," "gone away," or "house demolished," any other addresses of the patient or next of kin were tried. The response was good, but many remained uncontacted. Personal visits were next paid to all possible addresses. It was often found that the house had disappeared as a result of bombing, in which case the nearest intact dwelling was tried. By this means enough information was obtained to trace well over half the missing patients. Householders were almost without exception extremely co-operative, frequently going out of their way to gather information. Where they were unable to give exact details they could often give some help. For instance, the name of the road to which the patient had moved might be known, when reference to the voters' list would give the number. One patient was run to earth working in a public lavatory and another selling newspapers outside a well-known football ground. Some 400 miles of the streets of South London were covered in this part of the follow-up, which also included tracing a series of achlorhydric patients (in press). Where known and still practising, family doctors gave assistance, and for certain cases the medical superintendents and staff of other hospitals were of invaluable help. Local registrars of births and deaths and the Registrar-General provided details of dates and causes of deaths. In addition, particulars of any of the untraced patients who would now be aged over 50 were sent to the latter in case they had died and were identifiable in the records. Insurance House and the Central Pensions Branch of the Ministry of National Insurance were able to help in tracing some of the remainder.

Results of Follow-up—Both Series

The results of the follow-up investigations are given in Table IX.

TABLE IX.—*Results of Follow-up*

	Medical Series		Surgical Series	
	No.	%	No.	%
Alive and contacted	181	70·7	169	72·5
Dead	61	23·8	56	24·0
Alive, but not contacted	1	5·5	2	3·4
Emigrated and not contacted	3		2	
Nothing known	10		4	
Total	256		233	

Note: 6 patients in the medical and 21 in the surgical series died during their initial stay in hospital (see above) and are not included in the late analysis.

Deaths

Sixty-one patients in the medical and 56 in the surgical series have died since their initial stay in hospital. The causes of death in these cases are shown in Table X.

TABLE X.—*Causes of Death*

Cause	Medical Series		Surgical Series	
	Men	Women	Men	Women
A. Gastric:				
Carcinoma	2	1	3	1
Ulcer haemorrhage	4	1	—	—
Ulcer perforation	1	—	—	—
Ulcer—unspecified	1	—	—	—
Late post-operative	—	—	1	2
B. Other alimentary:				
Carcinoma large bowel	3	—	1	—
Appendicitis	1	—	—	—
Toxic hepatitis	—	—	1	—
C. Respiratory:				
Acute infections	7	1	7	—
Chronic bronchitis	1	1	4	—
Tuberculosis	3	—	6	—
Carcinoma of bronchus	3	1	3	—
Carcinoma of maxillary antrum	1	—	—	—
D. Cardiovascular:				
Heart disease	11	3	9	2
Cerebral vascular accidents	7	—	2	1
Syphilitic aortitis	—	—	1	—
E. Genito-urinary:				
Chronic nephritis	—	—	—	2
Hypernephroma	—	—	2	—
Post-prostatectomy	1	—	—	—
F. Metabolic:				
Diabetes mellitus	1	—	—	—
Addison's disease	—	—	—	1
G. Other:				
Basal-cell carcinoma	1	—	—	—
Bombing	—	2	—	—
Suicide	—	—	2	—
Unknown	2	1	4	1
Total	50	11	46	10

Note: Patients in the medical series who later had resections performed and who are not included in the surgical series have not been followed beyond the time of operation.

Despite the fact that the medical series might be expected to contain the majority of the aged and infirm patients, the death rates in the two series are almost exactly the same. No particular disease in either series predominates. There are too many factors involved for a more detailed statistical breakdown of the causes and rates of death to be of value, but two conditions, gastric carcinoma and pulmonary tuberculosis, require further analysis.

Gastric Carcinoma

Fifteen patients in the medical and 11 in the surgical series have developed carcinoma. In 10 (6 and 4 respectively) the primary growth was probably in the stomach. Brief particulars of these cases are presented in Table XI.

It has often been pointed out that the differentiation between chronic gastric ulcer and gastric carcinoma may be impossible before operation or even before microscopical examination of the specimen. The first two cases are almost certainly such examples, though, in both, operation was advised on account of the possibility of malignancy. In at least two of the remaining eight cases the carcinoma arose at a different site from the ulcer. It is interesting to note that in at least two, and possibly three, of the cases the removal of the large portion of the stomach, including the chronic gastric ulcer, did not prevent the subsequent development of a carcinoma.

Estimates of the incidence of malignant change in chronic gastric ulcers—"ulcer-cancer"—have come almost entirely from the pathologists, and very strict criteria for the diagnosis have been laid down, culminating in those of Newcomb (1932). If a certain percentage—for example, 3, 6, 10, or more—of gastric ulcers become malignant, it is remarkable that in none of the present cases do we find macroscopic or microscopical evidence of "ulcer-cancer." In the medical series alone the 262 patients have had some 2,000–3,000 years of ulcer history. When may we expect them to become malignant and show the features of "ulcer-cancer"?

Brinton (1864) described ulcerating carcinomata in which ulceration kept pace with growth for many years. According to Palmer and Humphreys (1944) such cases may show all the features of "ulcer-cancer." In a previous study of patients with gastric carcinoma (Swynnerton and Truelove, 1951) the evidence suggested that the histological diagnosis of "ulcer-cancer" was not always supported by the clinical facts. The problem of malignant degeneration of gastric

TABLE XI.—*Cases in Which there was Evidence of Gastric Carcinoma*

Case No.	Sex	Age	Length of History	Gastroscopy	Remarks
			Medical Series		
1	M	55	4 years	Irregular ulcer high lesser curve; ? carcinoma. Failed to heal	Refused operation. Died 19 months later of "carcinoma of stomach." No necropsy
2	M	57	6 ,,	Antral ulcer. Only one examination	Refused operation. Developed ascites. Died 1 month later of "carcinomatosis." No necropsy
3	F	74	50 ,,	High posterior lesser curve ulcer. Soundly healed after 5 weeks' treatment	Died 6 years later of "haematemesis due to gastric carcinoma." Before death had lost weight and became deeply jaundiced with enlarged liver. No necropsy
4	M	56	9 ,,	Ulcer at angulus. Healing 2 weeks later	2 years symptom-free, then recurrence lasting 1 year before admission with epigastric mass. Resection of prepyloric carcinoma. No evidence noted of preceding ulcer
5	M	53	16 ,,	Ulcer at angulus. Only one examination	Readmitted 6½ years later with gastric retention. At operation small localized prepyloric carcinoma, separate from old ulcer scar at angulus
6	M	51	1 week	Mid. anterior lesser curve ulcer. Soundly healed in 6 weeks	4 years symptom-free, then 1 year recurrence of dyspepsia before resection of high ulcerating diffuse spheroidal-cell carcinoma
			Surgical Series		
7	M	51	4 years	High posterior lesser curve ulcer	Partial gastrectomy. Histologically chronic peptic ulcer. Readmitted 9 years later with 2 years periodic dysphagia and substernal pain. Carcinoma of cardia resected
8	M	47	13 ,,	Ulcer at angulus	Partial gastrectomy. Histologically chronic peptic ulcer. Well for 6 years, then pain and diarrhoea for 4 months before death. Necropsy—diffuse carcinoma of stomach remnant
9	M	70	2 ,,	High lesser curve ulcer	A huge gastric ulcer penetrating and fixed to the liver treated by gastro-jejunostomy. Recurrent dyspepsia for 5 years, when he was admitted with an epigastric mass and nodular liver. Died—no necropsy
10	F	54	2 ,,	Low lesser curve ulcer	Partial gastrectomy. Histologically chronic peptic ulcer. 3½ years later admitted elsewhere with Krukenberg tumour of ovary and generalized abdominal carcinomatosis. Primary ? stomach. Died 8 months later. No necropsy

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ulcers is a clinical as well as a pathological one, and there is all too little clinical evidence in the literature. If there is an association between gastric ulcer and carcinoma, it seems to us to be due to a preference of both for the same soil rather than any direct association.

Pulmonary Tuberculosis

Pulvertaft (1952) has suggested that there may be an increased incidence of pulmonary tuberculosis following gastric resection of peptic ulcer. Among the 233 cases in the present surgical series six men have died of this disease —one seven months, three three years, one five years, and the other eight years after operation at the ages of 52, 49, 58, 62, 56, and 63 respectively. In none was the diagnosis recorded at the time of operation, but two patients who had active pulmonary lesions when operated on are still alive after seven and nine years, and their lesions are reported to be inactive. One woman died of Addison's disease 10 years after operation, and another was diagnosed as suffering from pulmonary tuberculosis seven years after gastrectomy.

In the medical series three men have died of pulmonary tuberculosis, in one only of whom was the diagnosis recorded at the time of admission for gastric treatment. The evidence concerning an increased risk of pulmonary tuberculosis following gastrectomy is inconclusive.

Manifest Haemorrhage

At least 139 (27.9%) of the 498 patients have had at some time haematemesis or melaena during the course of their ulcer career, and, of these, 22 (15.8%) have bled on more than one occasion.

Medical Series—Remaining Patients

After subtracting those patients who have died, those who have developed gastric carcinoma, and those who have been lost, there remain 178 on which to base the late results of medical treatment (Table XII). Transient gastric symptoms have not been considered sufficient evidence of recurrence. There must have been symptoms suggestive of gastric ulcer lasting for at least several days at a time before a diagnosis of recurrence was made. Occasional recurrences have been defined as those occurring less frequently than once a year. The seven patients who have died as a direct result of the ulcer are not considered further, since there is no adequate information on the subsequent gastric history in those who died from other causes for comparison.

TABLE XII.—*Late Results in the Medical Series*

	Men		Women		Total	
	No.	%	No.	%	No.	%
Recurrence requiring gastrectomy	46	36·5	11	21·2	57	32·0
Frequent recurrences	28	22·2	16	30·8	44	24·7
Occasional recurrences	23	18·3	12	23·1	35	19·7
No recurrences	29	23·0	13	25·0	42	23·6
Total	126		52		178	

In three-quarters of the patients the symptoms of gastric ulcer have recurred despite the fact that these cases might be expected to be mild or of recent onset. The large number of deaths should by the end of five to twelve years have eliminated the majority of patients who were not operated on initially because of age or infirmity. Re-examination of the criteria for inclusion of patients in this series does not reveal any obvious factor in the method of selection of cases to account for the appalling results, and one is forced to conclude that medical treatment, though it may heal the ulcer, will keep it healed in less than a quarter of the early or milder cases. These findings are in accord with those of Althausen (1949) for peptic ulcer as a whole.

The cases have been analysed to see if any factors influencing the recurrence rate can be found.

Age of Patient

That the age of the patient at the time of gastroscopic diagnosis is related to the incidence of recurrence is shown in Table XIII. There is a steady increase in freedom from recurrence with increasing age. There are several possible explanations of this finding, of which the most likely is that many of the patients whose ulcers recur frequently will have been operated on before they reach the age of 60. In addition, the older patients will be able to rest and relax more and to pay more attention to diet, etc.

TABLE XIII.—*Recurrence Rate in Relation to Age of Patient*

Age Group (years)	Recurrences					
	None		Occasional		Frequent	
	No.	%	No.	%	No.	%
Under 50	12	15·2	16	20·3	51	64·6
50–59	16	28·6	11	19·6	29	51·8
60+	14	32·6	8	18·6	21	48·8

Length of History

It might be expected that those patients with short histories prior to diagnosis would respond well to treatment and relapse rarely. Table XIV indicates that although they appear to do better than those with longer histories, their chances of remaining symptom-free are still under 50%. The numbers when divided into groups are small, but with a history of between one and twenty years the chances of prolonged freedom are only about one in eight. The results have been analysed in relation to both length of history and the age of the patient. Numbers are too few to yield significant results, but the trend is for patients under the age of 50 to have a higher incidence of recurrences than those over 60, no matter what the length of history. This, as mentioned above, may well be because those patients whose ulcers recur frequently are operated on before they reach an advanced age.

TABLE XIV.—*Recurrence Rate in Relation to Length of History*

Length of History (years)	Recurrences					
	None		Occasional		Frequent	
	No.	%	No.	%	No.	%
Under 1	14	40·0	7	20·0	14	40·0
1—	5	9·3	13	24·1	36	66·7
5—	6	23·1	5	19·2	15	57·7
10—	3	11·1	6	22·2	18	66·7
20+	11	36·7	4	13·3	15	50·0

Note: In 6 patients the length of history was unknown.

Diet and Alkalis

No coherent picture emerges from a study of the relationship of dieting and taking alkalis to recurrences. Some diet the whole time, some when they have pain, and the remainder never. The position may be summarized by saying that exactly 75% of those who have had no further recurrences have given up dieting, while 61.4% of those with frequent recurrences have stuck to a diet the whole time. Nearly all patients, whether they have had recurrences or not, try to avoid certain foodstuffs or other factors. Fatty foods, including fried fish, chipped potatoes, pastry, bacon, and pork are eschewed by the great majority. Other factors considered by the patients to be a cause of relapse are pickles, condiments, highly seasoned foods, tobacco, worry, and irregular meals. Since most of these are advised against in their diet sheets it is not possible to say whether they avoid them from personal experience.

The position of alkalis is even more involved. Few patients appear to take them prophylactically, but nearly all list them as giving the most immediate relief when symptoms are present. "Doctor's medicine" is usually the chief stand-by, but most take proprietary brands as well, frequently changing from one to another. Even patients who

state that they have had no further trouble keep alkalis to hand. Rest, milk, diet and regular meals, olive oil, liquid paraffin, and belladonna in various combinations were popular for ending attacks.

Most of the replies to the question of what gave the most relief were orthodox, but one man treated his recurrences by starvation, chlorodyne, and 3 pints of beer, another recommended stout, and a woman found that a little whisky taken medicinally was an excellent remedy. One patient had been "cured" by writing to a faith-healer, and two had obtained long remissions from an expensive medicine obtained from a pharmacist "specializing" in stomach troubles.

Surgical Series—Late Results

The late results in the 169 traced survivors have been assessed according to the grading of Visick (1948), which is as follows:

Grade I.—No gastric symptoms (fullness after an extra large meal is allowed).

Grade II.—No pain, mild occasional symptoms easily controlled by care. Care implies limitation of size of meal, rest, and avoidance of certain articles of diet.

Grade III.—Mild symptoms not controlled by care, slight abdominal discomfort. This grade is subdivided as follows: Grade IIIs, satisfactory (both the investigator and the patient are satisfied with the result; symptoms do not interfere with the enjoyment of life or with work); and Grade IIIu, unsatisfactory (either the investigator or the patient is not satisfied with the result; either the symptoms or the care taken to avoid them interferes with the patient's enjoyment of life or his efficiency at work).

Grade IV.—Not improved, severe symptoms.

The results in the present series are presented in Table XV.

TABLE XV.—*Chronic Gastric Ulcer: Surgical Series—Late Results*

Grading	Men		Women		Total	
	No.	%	No.	%	No.	%
Grade I	74	52·9	11	37·9	85	50·3
Grade II	38	27·1	12	41·4	50	29·6
Grade IIIs	14	10·0	3	10·3	17	10·1
Grade IIIu	9	6·4	3	10·3	12	7·1
Grade IV	5	3·6	—		5	3·0

Out of every 10 patients still alive the result has been satisfactory in 8, moderately successful in 1, and poor in 1. There is a small late operative morbidity which must be taken into account when judging the late operative results. One man died five years after partial gastrectomy following reduction of a retrograde jejunal intussusception, one woman two months afterwards following division of bands causing obstruction, and another four years later following operative procedures designed to relieve "dumping." A further three patients have been successfully operated on for obstruction due to adhesions. None of the unsatisfactory results were due to cases of recurrent ulceration following partial gastrectomy. Indeed, despite careful investigation of many post-operative cases in these years, we have been unable to find any case of proved stomal or gastric ulcer following gastrectomy for gastric ulcer. There have been several cases of iron-deficiency anaemia which have responded well to iron. In one case megaloblastic anaemia was given as a contributory cause of death. No patient has been encountered with any gross nutritional deficiencies, although many are below their best pre-operative weight.

Analysis of the late results to determine whether there are any specific factors influencing them shows that amongst the five patients in Grade IV three suffer from severe chronic bronchitis, one from angina pectoris, and the other, in whom there is no obvious associated disease, has been relieved, at any rate temporarily, since the start of this investigation by the type of gastric suspension operation described by Capper and Butler (1951). Of the twelve in Grade IIIu three suffer from chronic bronchitis, two from cardiac disease, another from chronic sciatic pain, and a seventh from osteoarthritis. On the evidence it would seem that the disgruntled chronic invalid is intolerant of any post-gastrectomy disability or discomfort.

The age of the patient at the time of operation makes little difference to the late results so far as gastric symptoms are concerned (Table XVI). Neither does the length of history of symptoms before operation affect the results to any significant degree.

TABLE XVI.—*Late Results in Relation to Age of Patient. Surgical Series*

Age at Time of Operation	Grades I, II, and IIIs		Grades IIIu and IV	
	No.	%	No.	%
Under 40 years	30	93·75	2	6·25
40— "	43	86·0	7	14·0
50— "	58	89·2	7	10·8
60+ "	21	95·5	1	4·5

Comparison of Late Results

Symptoms due to chronic gastric ulcer tend to be intermittent while the non-ulcer complications following surgery tend to be constant, so no exact comparison of the symptoms in the two series is possible. If Visick Grades I and II are taken as being equivalent to no recurrences in the medical series, Grade IIIs as occasional recurrences, and Grades IIIu and IV as frequent recurrences the results shown in Table XVII are obtained.

TABLE XVII.—*Chronic Gastric Ulcer: Comparison of Results in the Medical and Surgical Series*

	Medical Series		Surgical Series	
	No.	%	No.	%
"No" recurrences ..	42	23·6	135	79·9
"Occasional" " ..	35	19·7	17	10·1
"Frequent" " ..	101	56·7	17	10·1
Total	178		169	

Only a quarter of the patients in the medical series, compared with over three-quarters in the surgical series, have had no further trouble. This despite the probability that the medical series contained the majority of patients whose ulcers were initially considered to be mild or of short duration.

Discussion

Absolute indications for surgery are perforation, certain cases of manifest haemorrhage, obstruction due to hour-glass deformity or pyloric stenosis, and suspicion of malignancy, which includes all ulcers on the greater curve and the majority in the pre-pyloric region. Elective surgery is advisable when there is failure to respond to medical treatment or recurrence after a previous course or courses of strict treatment.

The analysis shows that partial gastrectomy is by far the most satisfactory method available at present for the treatment of chronic gastric ulcer. Before it is advocated various factors must be considered. These include the length of history and severity of symptoms, the presence of intercurrent disease, and the operative mortalities. Early operative mortalities will depend to a large extent on local factors, but they have significantly diminished in most parts of the world since the earlier part of this series. Late operative deaths and complications will probably be fairly constant wherever the operation is performed. In the present series the late operative complications are more than counterbalanced by the late deaths directly due to the ulcer in the medical series.

On the figures in the present analysis a strong case can be made out for surgery as soon as the diagnosis is arrived at, except in those patients with very short histories and those with signs of senility or intercurrent disease, especially severe chronic bronchitis. There are few who would be prepared to operate on all such cases, and a full course of medical treatment will relieve a small proportion for at

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least a number of years. Recurrence after a strict course of medical treatment should be regarded as an indication for surgery, whether or not a strict post-ulcer regime has been followed.

Summary

The case records of 498 patients with chronic gastric ulcer who were treated in the gastro-enterological clinic at St. James's Hospital, London, during the years 1940–6 inclusive are analysed. Adequate follow-up data concerning nearly all were obtained in 1952.

The patients fall into two series, the one treated medically and the other surgically. In the former there are 262 and in the latter 254 patients, 18 appearing in both series.

The criterion for inclusion was the presence of an active chronic gastric ulcer, which in the medical series must have been seen gastroscopically. Patients with additional duodenal ulcers or scars have been excluded.

The ratio of men to women was 3.5:1.

The symptoms may start at any age, most commonly after 30, the average age being 44. The average age at the time of operation in those surgically treated was in the fifties.

The sites of gastric ulcers differ greatly between men and women. In women the site of predilection is the high posterior aspect of the lesser curve, and ulcers in the pyloric antrum are exceptional. In men the distribution along the lesser curve is more even, with a maximum incidence in the mid and lower body, while 10% are situated distal to the angulus.

The results of a fractional test meal, using gruel as the stimulant, are given. The age of the patient influences the level of acidity to only a slight extent, and it is concluded that the presence of a degenerate mucosa rather than the age of the patient accounts for the findings.

A quarter of the patients in each series have subsequently died. There is no predominant cause of death.

The problem of the malignant degeneration of chronic gastric ulcers is considered. In none of those who later developed gastric carcinoma is there enough evidence to label them "ulcer-cancers," and it is concluded that if an association between gastric ulcer and gastric carcinoma exists it is due to a preference of both for the same type of degenerate mucosa.

The evidence concerning the incidence of tuberculosis following gastric resection is presented. No conclusion concerning this is reached.

In 28% of patients an incident of manifest bleeding occurred during the course of their ulcer histories. In 16% of these there was more than one episode.

In the medical series it is concluded that although treatment will nearly always heal the ulcer it will keep it healed in only a quarter of the milder or earlier cases. A quarter have symptomatic recurrences more often than once a year, and a further third have had subsequent resections performed for recurrence. Some of the factors which may influence the recurrence rates are analysed.

Three-quarters of those who have had no further trouble have given up dieting, while nearly two-thirds of those with frequent trouble have kept to a diet the whole time.

In the surgical series a very satisfactory result has been obtained in 80%, a moderately successful one in 10%, and a poor result in the remaining 10%.

A high proportion of those with poor results have some other chronic incapacitating disease, most frequently chronic bronchitis.

The choice of treatment for those presenting at hospital with a chronic gastric ulcer is discussed. It is concluded that surgery at present offers the best results, but before it is advocated certain factors, including local operative mortality figures, the severity and length of history of symptoms, and the presence of intercurrent disease, should be taken into account.

Mr. Colin Craig has very kindly allowed us to make use of his late results for patients operated on in 1946. We wish to thank the many people who co-operated in the follow-up of patients, and are grateful to the staff of St. James's Hospital for their interest and advice. We are particularly indebted to Miss Kirwin, who, in addition to secretarial duties, suggested ways and means which led to the tracing of many "lost" patients. The cost of the investigation was met by a grant from the Weir Research Fund.

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CANCER AND THE COMMUNITY*

BY

PERCY STOCKS, C.M.G., M.D., F.R.C.P.

Cheshire and North Wales Branch, British Empire Cancer Campaign

In considering the scope of this discussion I have assumed that the Section of Preventive Medicine is not directly concerned with treatment, except in respect of diseases whose spread in the community can be reduced by early treatment of patients infective to others. This absolves me from dealing with the rather controversial questions of how far the community might be influenced to seek medical advice at an earlier stage of cancer and what effect that could have upon ultimate survival. Those aspects of the problem are important to the community and interesting to statisticians, whether in Toronto or London, but, while they may affect the fatality of cancer, they cannot, so far as we know at present, affect the incidence or prevention of the disease.

Attitudes to the Cancer Problem

Some will say that since we can do so little as yet to prevent cancer it is unrealistic to discuss it at all in this Section. Such an attitude seems generally to stem from a fixed idea that cancer is a complication of growing old, or a mode of departing from this life, which is inevitable for a proportion of people in the same sense that arteriosclerosis is inevitable, and I propose to deal with some of the misconceptions mixed up with that idea. According to my observations it is the medical people rather than the lay community who tend to the cheerless view that the best we can do is to try to

*Read in opening a discussion in the Section of Preventive Medicine at the Annual Meeting of the British Medical Association, Cardiff, 1953.

improve the effectiveness of treatment, and that it is futile to try to discover causes of cancer which would be capable of elimination. However, if doctors are at all happy about advances in the treatment of this disease the community certainly is not, and I sense a growing feeling of doubt whether the ordeal of treatment is worth while in many cases in order to achieve a short prolongation of life, coupled with some surprise and impatience because so little serious attempt seems to be made, apart from work on animals, to discover causes with a view to abating them. I find, for example, that, whilst only a very small percentage of people decline to furnish details about their environmental histories, many doctors are hesitant, for one reason or another, in furthering this type of research.

It is sometimes said, generally by young doctors, that cancer is of comparatively slight economic importance to the community, since it is predominantly a disease of advanced life. Comparison with tuberculosis a decade or two ago gave some justification for the view, but it is not true to-day, and it is time that error was exposed. In an address to the British Tuberculosis Association in 1948 (*Tubercle*, 1949, **30**, 50) I calculated the years of future "working" life between the ages of 15 and 65 lost to the community in England and Wales through premature deaths caused by respiratory tuberculosis, cancer, and accidents in 1945. It was assumed that if the people who died of one of those causes had not done so in that year, they would have been subject from that time onwards to the ordinary risks of mortality at successive age periods as shown by the 1945 life table rates for the whole population of each sex. The future working years lost as a result of the cancer deaths was 355,000, compared with 435,000 lost through deaths by respiratory tuberculosis and 268,000 lost by fatal accidents. In view of the great fall in tuberculosis mortality since 1945 I have now repeated the whole calculation for the year 1950, using the life table for that year, and the result is shown in Table I. No account is taken of the loss through invalidity prior to death.

TABLE I.—*Estimated Loss of Future Working Life Due to Cancer, Tuberculosis, and Accidents in* 1950

Cause of Death in 1950	International Numbers	No. of Thousands of Person-years of Future Life Lost between 15 and 65		
		Males	Females	Total
Cancer	140–205	212	220	432
Tuberculosis, respiratory	001–008	143	125	268
Accidents	E800–962	211	61	272

The cancer total has increased from 355 to 432 thousands in the interval because the expectation of life based on 1950 death rates from all causes was much better at each age than in 1945, and because the new definition of cancer takes in leukaemia and Hodgkin's disease; this does not indicate a worsening of cancer mortality for both sexes taken together. For accidents the figure has hardly changed, owing to a balance between the reduction in deaths and the increase in normal life expectation. Accidents and cancer are now equally important for males according to this criterion, but for females cancer produces three and a half times the loss due to accidents. For respiratory tuberculosis there has been a great fall from 435 to 268 thousands, bringing the total much below that for cancer. Consequently, cancer has now become the cause of death which is of most serious economic importance when judged from this standpoint. It is interesting to note that cancer of the lung and bronchus in 1950 contributed 65 to the 212 total for male cancer deaths, and this figure is of course increasing rapidly every year. Cancer of the stomach contributed 33 thousand years for males, so nearly half the total is due to these two forms of cancer in the case of men.

Possibilities of Reducing Cancer Incidence

Another prevalent idea among doctors, though not in the community as a whole, is that it is of little profit to talk about abatement of cancer until pathologists have established its aetiology through experiment, despite the fact that this thesis has been disproved in the history of other diseases. I was once asked by a group of demographers why we could be any more confident in predicting the future trend of the general death rates at particular ages than we are in predicting the trend of the birth rate. My reply was that whilst the latter is influenced by many conflicting desires and considerations which pull in opposing directions and are affected by the social circumstances of the time, conscious efforts to influence mortality in a free democracy are pulling all the time in the same direction, downwards, and, apart from wars, we can count on that continuing.

Furthermore, experience has shown that when the public conscience has become thoroughly aroused about deaths from a particular cause, that death rate has tended to come down whether or not the pathology was properly understood. Two decades ago no one thought that the national infant mortality rate would ever be reduced below 30 per 1,000; but world-wide interest in the problem accompanied by all-round efforts without much aid from new discoveries resulted in rates of 20 being attained by Sweden and 25 by Holland in 1950, and brought down the England and Wales rate to about 28 in 1952. About 1934 the public conscience became thoroughly aroused about maternal mortality, and the rate began to fall at once and continued to do so for years before the sulphonamides and penicillin came along and nearly finished the job. The trouble with cancer is that possibilities of abating its incidence have as yet hardly been regarded seriously except in respect of a few varieties of so-called industrial cancer whose causes are well established.

The search for connexions between human cancer and the countless chemical substances, natural and artificial, which we ingest, inhale, and absorb in the course of our normal lives has begun, however, and we really owe its inception to the observation of Percivall Pott, a surgeon at Bart's in the eighteenth century, that skin cancers of chimneysweeps arose from irritation by soot; there followed, more than a century later, proof that certain mineral oils were responsible for the epitheliomas occurring in cotton-spinners.

Chemical Promoters and Irritants

The number of different agents which have been proved to be carcinogenic to animals since Yamagiwa and Ichikawa first produced true malignant tumours by applying coal tar to the ears of rabbits in 1915 now runs into many hundreds. In 1922 Passey produced malignant growths by painting the skin of mice with an ethereal extract of soot, and in 1924–5 Kennaway was able to produce carcinogenic tars by methods which led to the conclusion that the carcinogenic agent was a complex hydrocarbon of the aromatic series. In 1927 Mayneord discovered that the carcinogenic tars and oils had a characteristic fluorescence spectrum, and Heiger then found that a chemical substance, 1:2-benzanthracene, gave such a spectrum. As a result of further intensive research Kennaway and Heiger were able to demonstrate for the first time that cancer could be produced by a pure compound 1:2:5:6-dibenzanthracene, and in 1933 Cook and Hewett synthesized 3:4-benzpyrene, a carcinogen present in pitch. More recently Berenblum and others have produced experimental evidence that after application of a

carcinogen certain non-specific irritants could determine whether or not tumours appeared and how long they took to appear.

Furthermore, it has been found that the addition of certain fats to the diet of experimental animals increases the incidence of tumours resulting from application of some carcinogens to the skin, whilst, on the other hand, hydrogenated coconut oil reduces the incidence. This work on non-specific co-carcinogens and anti-carcinogens might possibly open the way to cancer prevention on a large scale without waiting until the whole mechanism is understood. Consider the case of bladder cancer, which we know is produced with rather high frequency in men who work with certain chemicals of precisely known composition, while thousands of people who have never handled those chemicals industrially contract the same condition. Surely it is a reasonable inference that those other bladder cancers are initiated by contact in ordinary life with chemicals closely related to the known industrial carcinogens, and whether they do develop may depend on the action of some other chronic irritant. This form of cancer becomes, therefore, a problem for preventive medicine to investigate and is not merely a problem peculiar to one industry.

Statistical Research on Cancer

A third manifestation of the sceptical attitude to prevention in respect of cancer is the excuse that statistics cannot prove causation, and, since they can be made to prove almost anything, why take any notice of them? When statistics show a death rate to be falling the evidence is usually accepted without question and credit is taken for it by all who can lay claim to a part in that happy event. But if statistics show a rate to be rising for a particular disease it is at once objected that medical certification or changing fashion in nomenclature made the figures fallacious. The persistence of the old joke about statistics proving anything is convenient, because when statisticians arrive at unpalatable conclusions it can be said that so long as the evidence is solely statistical nothing need be done except wait for "real," that is to say experimental, evidence. If many thousands of people are dying annually from a disease and statistical evidence indicates that the probability is very high that the main cause lies in an article X, it is hardly sensible to sit back and wait, maybe 20 years, for the experimental evidence. Indeed, unequivocal evidence may never be obtained in a case of a disease which takes a very long time to develop in man after application of an irritant or of an initiator. In such a case the responsibility of advising the community is a serious one, and it is idle to suppose it can be evaded by pretending that statistical evidence alone is of no account.

We are faced with such a situation to-day in regard to lung cancer and tobacco-smoking, and, whilst it is fortunate that tobacco is not a necessity of life, the position is complicated by considerations of finance. In my opinion the proper course is to intensify research on the question from every aspect, and meanwhile inform the community of the statistical findings (as indeed has already been done by the B.B.C.), and say what the risk amounts to if what is suspected is correct, so that each individual can decide for himself or herself whether to take the risk or not. Although statistical studies alone may establish the existence of a real connexion between a factor X and a disease Y without revealing the precise reason for that connexion, the probability that in the absence of X there could be a great reduction in Y may be indicated as so high that some may consider that the use of X involves an amount of risk which it is unwise to take.

Whatever may be the final outcome of the tobacco question, cancer of the lung has now come into the field of preventive medicine. Non-smokers can get the disease and the statistics of deaths suggest that smoke from domestic chimneys may be a factor, so the problem to be solved is whether it arises through inhaling or ingesting carcinogenic or co-carcinogenic substances derived from coal or mineral oil or used in the cultivation or manufacture of tobacco. Perhaps an initiator is present in one of these and a non-specific irritant in another, and statistical studies now in progress may help to answer this question.

Cancers of the oesophagus and larynx are more frequent amongst men in occupations associated with excessive drinking of alcohol, but there are other factors involved, and the sexes are differently affected. Cancers of the cervix uteri on the one hand and of the breast and ovary on the other are associated in opposite ways with marital history. For these and other kinds of cancer, statistics have indicated that environmental factors have some influence on incidence.

Incidence of Cancer of the Stomach

In the case of stomach cancer incidence is closely related to social and urban environment, and it shows other peculiar local variations for which no explanation has yet been found. Death rates in 1921–30 attributed to this cause showed considerable excess in London and aggregates of large towns at early ages, but the excess was not so pronounced after 65. Standardized comparative figures for years 1947–9, based upon the mortality expected by applying age-specific rates for all rural districts of England and Wales to the populations, are shown in Table II.

TABLE II.—*Comparative Mortality Figures for Cancer of Stomach in Certain Large Towns in 1947–9 (With Rates for All Rural Districts of England and Wales as Base)*

	Males			Females		
	15–	45–	65+	15–	45–	65+
Liverpool ..	185	156	144	175	128	131
Manchester ..	218	158	132	124	151	123
London county ..	155	114	101	135	109	106
Outer ring of Greater London	133	97	97	112	91	100
Birmingham ..	130	125	120	155	125	116
All rural districts	100	100	100	100	100	100

There has been no diminution of the urban excess previously noted. It is very pronounced in Liverpool and Manchester, and is also considerable in Birmingham, at every age amongst both men and women. In London the excess is evident only at the younger ages, and rates are higher at each age in the centre than in the outer ring. This suggests that some carcinogenic or co-carcinogenic substance, which might be ingested or inhaled in the first instance, is more often encountered in large towns than in rural areas; but we also know that certain rural areas present a large and at present inexplicable excess of mortality compared with other rural districts. I refer particularly to the counties of Wales which touch the sea coast, and will confine attention to the five in North Wales which are now being studied by the Cheshire and North Wales Branch of the British Empire Cancer Campaign with the help of the medical officers of health, the Liverpool and Welsh Regional Hospital Boards, and the University College of North Wales. Comparative indices for cancer of the stomach and breast in North Wales during the 29 years from 1921 to 1949, based as before on the mortality expected from rates in all rural districts at decennial age groups, are shown in Table III. The North Wales counties included are Anglesey, Caernarvon, Denbigh, Flint, and Merioneth; the West Midland group comprises Gloucester, Stafford, Warwick, and Worcester; and the South-east group consists of Kent, Surrey, Sussex East, and Sussex West. In this table, and also in Table II, duodenum is included with stomach.

North Wales showed a rural mortality attributed to stomach cancer about twice the national average at ages under 65, and about 1.8 times the average at ages over 65. Dividing into two periods 1921–39 and 1940–9, the excess is seen to have become only slightly less in the second than in the first period. Cheshire and Lancashire rural districts produced average rates, whilst the West Midland rates were about 11% below the national level, and the South-east

TABLE III.—*Comparative Mortality Figures for Cancer of Stomach and Breast in 1921–49 in Aggregates of Rural Districts of North Wales and Parts of England (With Rates for All Rural Districts as Base)*

Rural Districts of	Stomach				Breast	
	Males		Females		Females	
	15–	65+	15–	65+	15–	65+
North Wales 1921–39	184	173	224	203	95	91
North Wales 1940–49	177	165	210	173	97	95
North Wales 1921–49	182	169	220	192	96	93
Cheshire and Lancashire, 1921–49	101	97	99	104	95	101
West Midlands, 1921–49	89	89	86	89	105	106
South-east, 1921–49	81	83	73	76	105	100
All England and Wales	100	100	100	100	100	100

county rates were around 20% below. By way of contrast, breast cancer mortality in the 29 years showed no important differences between these areas, that in North Wales being 5% below average, and that in the West Midlands 5% above.

Justification for Research into Environmental Factors

Suggestions have been made that doctors in Wales tend to certify cancer of the stomach as the cause of death whenever haematemesis or obscure gastric symptoms make it seem feasible, and often with insufficient justification. No evidence that such a practice is in fact more prevalent in Welsh than in English rural areas has been produced. The relative excess is smaller at ages over 65 than in mid-life; and no compensating deficiency can be found in the group of deaths attributed to those conditions likely to be mistaken for gastric cancer, such as peptic ulcer, cancer of the liver, pancreas, and colon, other diseases of the stomach, and pernicious anaemia. Inquiries from a first sample of 100 certifiers of deaths said to be due to stomach cancer in North Wales show that there was necropsy, biopsy, operation, or *x*-ray evidence for 50%, compared with 65% of 100 deaths so certified in rural districts of Cheshire and Lancashire, and about 60% in the large towns other than Liverpool. If half the cases in which the diagnosis is unsupported by any of these tests are rejected, the North Wales rates would be reduced by a quarter and the Cheshire–Lancashire rates by a sixth, correcting the ratio between them to 1.8 at ages under 65 and 1.6 at ages over 65.

These preliminary statistical tests justify the Cheshire and North Wales Branch of the British Empire Cancer Campaign in their belief that there exists a real phenomenon to be explained, and in their determination to investigate race and residence, work and workplace, soil, air, and water, food, drink, and every conceivable vehicle of carcinogenic substances, or co-carcinogenic irritants. With this line of approach the community of the research area seem to be very much in sympathy. It is something they can understand and welcome because it holds out some hope of prevention, which from the patients' standpoint would be so much better than cure by surgery or radiation, no matter how skilful or high-powered that may be.

Contribution of Medical Officers of Health to Environmental Studies

At the International Conference in London in 1928, organized by the British Empire Cancer Campaign, Sir George Buchanan gave an address on "Collective Public Effort in Dealing with Cancer," which after 25 years is still very pertinent to the discussion to-day. Speaking of municipal work, he said: "The principle to be followed . . . is to secure the closest possible common action between the municipal health department and the authorities of the general and poor-law hospitals, the university if there is one, the voluntary agencies which support social work, and especially the staffs of hospitals and the medical practitioners as well as the local work of the B.E.C.C. where it is in operation. It is no part of the programme that municipal direction should be made prominent; it is enough for the health department if it can help. Where the medical officer of health is wanted to take the initiative he will do so; otherwise he is content to be one of the cogs on a useful working wheel. Personally, I may express the view that cancer research has much to gain through his instrumentality. . . ."

I would like to take this opportunity of expressing my profound appreciation of the way in which the medical officers of health of the region in which I am working are, together with their assistants, helping the British Empire Cancer Campaign in just that spirit.

Sir John Robertson, at that time medical officer of health of Birmingham; uttered at the same conference some words on which I will close. "Inquiry by the health authority into the facts surrounding each death from cancer has as much value from an educational point of view as it has from an aetiological." How true this is we are finding out in the study I have referred to; education of surgeons, physicians, radiologists, and health officers about the essential gravity of cancer as a community problem is proceeding as a result of the inquiries in progress, and later it may even penetrate to the university. Whether the scientific results we hope for materialize or not, by such education the community will benefit eventually.

CANCER AND THE COMMUNITY*

BY

RONALD W. RAVEN, O.B.E., F.R.C.S.

Surgeon to Westminster (Gordon) Hospital; Surgeon to the Royal Cancer Hospital

The control of cancer is a world problem and one of the big challenges of the present day. Its magnitude requires an all-out effort to reach a solution. With our present knowledge of the disease a great deal can be done to help, but we must not be satisfied until it is under complete control. The object of this paper is to indicate the effects of cancer on the community and the ways in which the latter can co-operate in the fight for its mastery.

Effects of Cancer on the Community

These are of such severity that the disease can be described as one of the greatest scourges of mankind.

World Mortality.—The death rate from cancer varies in different countries, and the figures shown in the Table were published by the World Health Organization in 1951.

Deaths from Cancer by Country per 100,000 *Population*

Country	Males	Females
United Kingdom (1950)	205·8	184·0
France (1950)	175·8	169·5
Northern Ireland (1951)	161·3	145·1
Netherlands (1951)	151·4	147·2
U.S.A. (1949)	141·1	136·4
Canada (1950)	129·7	123·1
Israel (1951)	85·5	105·5
Japan (1950)	80·1	74·9

National Mortality.—Reference to the Registrar-General's Statistical Review of England and Wales for 1950 showed that 87,274 persons died from neoplasms, as compared with 72,613 in 1940. During this decade the death rate has fallen considerably from acute infec-

*Read in opening a discussion in the Section of Preventive Medicine at the Annual Meeting of the British Medical Association, Cardiff, 1953.

tive and parasitic diseases, tuberculosis, rheumatism, and respiratory diseases as a result of early diagnosis and advances in treatment, in which antibiotics have played an important part. Workers in public health and preventive medicine have made a big contribution in these and other fields, but the cancer situation remains serious in that the number of deaths in this decade has risen by 14,661 and only cardiovascular disease has a higher national mortality. It is not without significance that the greatest number of deaths are caused by cancer of the alimentary tract, especially of the stomach and colon, and these are rising. If the two conveying tubes, the oesophagus and the rectum, are compared, deaths from cancer are nearly three times greater in the latter organ.

Family and Individual Problems.—About half the total deaths from cancer occur in persons between 45 and 65, when their family and business responsibilities are greatest. The large number of children who die annually from cancer and allied diseases should also be remembered. The development of cancer is one of the worst events that can overtake us, for it means either a major operation, a course of radiotherapy, or both, and subsequent rehabilitation may take a long time, with the necessary follow-up examinations at regular intervals. Life may be resumed with a colostomy or tracheostomy, and when the disease is more advanced plans must be made for a chronic illness with all its repercussions on the life of the family. These things are brought out in the report of the Joint National Survey Committee (1952), of which I was chairman.

The present situation regarding mortality and morbidity is serious, and everything possible must be done to rectify it.

Control of Cancer.—The joint endeavours of the clinical and laboratory workers which are applied to gain this final objective can be considered as a five-point programme. Every member of the community has a responsible part to play.

Research into Causation

Studies concerning animal cancer in the laboratories have brought to light a number of genetic, physical, and chemical factors which cause malignancy. They have also been identified with human cancer; for instance, in the development of certain occupational and industrial forms of the disease exogenous physical and chemical carcinogens are recognized. It is also possible that some spontaneous varieties of cancer may be caused by other unrecognized carcinogens, either exogenous or endogenous, for which a determined search is desirable. Clues might come from a careful scrutiny of the environment and habits of people of various nations, for it is known that the incidence of the disease varies with race, country, and economic status. A more rigorous search for carcinogenic substances should be made in the tissues of patients with various forms of cancer, and it must be remembered that their action may be additive.

There is scope for increasing the amount of clinical research in the wards and of study of carefully compiled clinical records of astute observers. Obvious advantages would be gained by drawing clinical and laboratory workers closer together so that they could discuss their problems and share ideas. It should not be overlooked that some of the fundamental facts in cancer research were contributed by clinicians; for example, our knowledge of chemical carcinogens was built upon the observation of Percivall Pott that cancer of the skin of the scrotum was common in chimney-sweeps. Fundamental planned research in the laboratories concerning the nature of the cancerous change in normal cells is a long-term project, but in the meantime there are many other problems to tackle whose solution may throw considerable light on the subject.

Prevention of Cancer

The community must recognize that certain varieties of cancer are preventable if the appropriate action is taken. Were further research to reveal that common types of the disease are caused by exogenous carcinogens it would be possible to gain considerable control by their elimination alone.

Precancerous Lesions.—A number of these conditions are well recognized, and more precise information is required regarding their frequency in the community and the degree of increased risk which they entail. These facts would be taken into account in the assessment of the need for cancer-detection centres where precancerous lesions could be dealt with. The association of this work with the prevention of cancer is obvious, and a few examples will suffice. A benign tumour such as an adenoma of the colon or rectum, a papilloma of the urinary bladder, or a duct papilloma or fibro-adenoma of the breast is carefully removed. If a benign melanoma is in a position where it might be injured it should be excised. Patients with familial polyposis of the colon and rectum develop cancer at a relatively early age, and the affected part should be removed before this occurs. Cancer is likely to develop in the colon affected by long-standing ulcerative colitis, so total colectomy is being performed more often in these patients. The association of oral sepsis, syphilis, and leucoplakia with cancer of the mouth has been recognized for a long time, and the incidence of this form of the disease will diminish by their timely treatment.

Cancer and Industry.—Workers in certain industries are exposed to carcinogenic substances which affect the skin and other parts of the body. The effects of mineral oils and tar are well known, and more recently benzidine and β-naphthylamine, used for making dyestuffs, have proved to be dangerous chemicals. Prevention of cancer in these industrial workers includes the isolation and separation of carcinogenic substances from the materials used by them, thus giving a complete safeguard. In the meantime the provision of protective appliances for the skin, adequate washing accommodation, good ventilation of factories, and the formation of regulations for the guidance of workers are of obvious importance.

Lung Cancer and Cigarettes.—As a result of the careful work by Doll and Bradford Hill we can say that cigarette-smoking is a potentially dangerous pursuit, and before starting the habit people should weigh up the increased risk of developing lung cancer in middle and later life. More investigations are necessary to see whether a carcinogen can be isolated from cigarette tobacco.

Lung Cancer and Atmosphere Pollution.—The high death rate from respiratory diseases in residents of the industrial centres of the country has been recognized for many years, and was emphasized during the great fog of London in the winter of 1952. It has been suggested that the continuous exposure of persons to a carcinogen in the atmosphere such as benzpyrene lowers the resistance threshold of the lung to cancer in cigarette smokers. More work is required concerning local atmospheric pollution in the neighbourhood of gasworks and its effects on the respiratory system.

Early Diagnosis and Treatment

The best chance for a patient with cancer lies in early diagnosis and efficient treatment, and my experience proves that it can be cured in many patients. The community has a special responsibility to report to the doctor when any disturbance in health occurs lasting more than a few days. Conditions other than cancer may be the cause of this, and by timely treatment they can be rectified. Some may hesitate to go to the doctor because they fear cancer, so by

wise teaching we must help them to conquer it. Progress is being made in the treatment of cancer, and people can take courage and find new hope. The present methods available include surgery, radiotherapy, chemotherapy, and combinations of these treatments.

Surgery, helped by advances in anaesthesia and antibiotics, has been extended to a number of organs, and radical operations for cancer of the oropharynx and hypopharynx, oesophagus, and lung can now be performed with a great measure of safety. Other operations have become more radical, thus increasing the percentage of operability and survival rates; these include resection of the stomach, colon, and rectum. Radiotherapy, alone or combined with surgery, has made a valuable contribution, especially in cancer of the skin, breast, cervix uteri, and larynx. Radioactive isotopes are helpful in cancer of the bladder and thyroid. Much work is also being done in chemotherapy to find substances to control cancer. Oestrogens retard the growth of cancer of the prostate or inactivate it; when the disease is advanced and the patient has been treated by castration and oestrogens, adrenalectomy may help further. A number of women with cancer of the breast are helped by the withdrawal of oestrogens by oophorectomy or the administration of androgens. I have known several of these patients with advanced cancer live normally for a number of years without any pain.

Rehabilitation of the Patients after Treatment

Following treatment the general condition of patients is usually lowered together with the ability to work. Hospital treatment is unnecessary, but they are not fit enough to go home; this interval must be bridged by convalescent treatment. Homes are therefore necessary for them which are staffed by those who are trained for this special work. The treatment includes rebuilding the patient's nutritional state, correcting any blood deficiency, restoring muscle tone and development. A colostomy or tracheostomy may be present, and patients may require instruction in their management. Following laryngectomy or laryngo-pharyngectomy speech therapy is necessary. Other patients may have an amputation stump which requires attention, together with instruction concerning wearing an artificial limb. Patients must also have an opportunity to reorientate their general outlook, and this programme of treatment requires several weeks to complete.

Care of Patients with Persistent Cancer

At present this is a big problem, but it is hoped that it will diminish in the future as a result of earlier diagnosis and treatment. Many of these patients are nursed at home and much help is required for them and their relatives. This was shown in the national survey report, where a description is given of how this can be done. Special assistance is available from organizations such as the Marie Curie Memorial Foundation and the National Society for Cancer Relief. There are homes where many can be given all the care and treatment required; some can go temporarily so that the family can have a rest. The maintenance of the general health, the nutritional state, and the condition of the blood are important in improving the patient's resistance to the disease. Modern methods for the relief of pain, palliative radiotherapy, and chemotherapy have proved valuable in certain circumstances. These patients often face a long illness, so care is taken to avoid despondency; much can be done to help them, and no impression must ever be given that treatment has ceased.

Cancer Education for the Public

It is clear that the community has its responsibilities in this programme for cancer control, chiefly in connexion with the prevention and early diagnosis of the disease. Before people can play their part they must be advised about the steps they can take under certain conditions in a way which will gain their confidence.

In 1804 an attempt was made to give people information on this subject, but the mails were so slow that the patients died before it reached them. Since that time a number of surgeons have stressed the need for this work. For example, in 1900, when Wertheim introduced his operation for radical removal of the uterus, a movement was started in Germany to educate women to recognize the early symptoms of cancer of the cervix uteri. The Marie Curie Memorial Foundation has a plan to carry out cancer education on a national scale. The object is to substitute intelligence and optimism for ignorance and fear. It is not enough to tell people what are the early symptoms of cancer; they should also be told how to act in certain circumstances and what can be done to help them. It is recognized that this must be done wisely and by stages until the whole population is eventually covered. An important approach to the public is through the medical officers of health, and already a number have set up education committees to deal with the work. These committees should be expanded to cover the country, and in the first place information given to specified groups, including the district nurses, who number 13,000 in England and Scotland; health visitors, numbering about 6,000; and student nurses, numbering 50,000. The education scheme should also embrace all the social and welfare workers, and by this means a nucleus of informed people will be created through whom the facts can reach the population. Expansion will occur by degrees by taking in other sections until the whole country is covered.

Methods of Teaching

Use of Leaflets.—A series of 12 leaflets setting out the early symptoms of cancer in the chief sites of the body, together with notes on its prevention and treatment, is available at the Marie Curie Memorial Foundation to medical officers of health and others on request for careful distribution.

Lectures and Films.—Groups of people are invited to lectures on cancer which are illustrated by lantern slides, and educational films are shown. Experience shows that these are well attended, and excellent discussions follow the lectures. Special lectures are also given to members of the nursing profession in refresher courses, and, in addition, arrangements are made for groups of district nurses to attend at a cancer clinic so that they can see the results of modern diagnosis and treatment. Talks on the radio can also be developed, and these will reach a large section of the community.

Publications.—Periodically, articles on various aspects of cancer are published for nurses and health visitors to help them in their work. In addition, simple articles published in the lay press are of value to the public.

Presentation.—The subject must be presented in simple language which is easily understood and designed to develop an optimistic outlook and confidence. A complete picture is given, including information on the nature of the disease, the way by which it may be prevented, the early symptoms and signs, and what can be done for the patients. The latter includes the help which is available from the various organizations working in this field.

Summary

The effects of cancer on the community are described with special reference to world and national mortality, together with family and individual problems. It is shown that the present situation is serious as regards mortality and morbidity. A five-point programme for the control of cancer is described and the part the community can play is shown. An outline of a scheme for cancer education of the public is given.

MODIFIED COURSE OF T. CONGOLENSE INFECTION IN MICE GIVEN DIETS WITH MILK CASEIN*

BY

AUDREY A. N. KEPPIE, B.Sc.

Bacteriology Department, University and Western Infirmary of Glasgow

In February, 1952, mice which had been fed on diets with different amounts of milk casein as protein were inoculated with *Trypanosoma congolense* (Busimbi strain). Contrary to expectation, blood films from the animals failed to reveal trypanosomes and the mice did not die. This phenomenon was so unexpected that it was further studied with a range of diets as described in this paper. The results of two further experiments confirmed the original observation and showed that the unexpected result might be attributed to a change of diet.

In the absence of fuller investigation the results were not published. However, since they show a close analogy with the findings of Maegraith, Deegan, and Jones (1952) of the suppression of *Plasmodium berghei* infection in rats fed on milk diets, it now seems desirable to give a brief account of the experiments with trypanosomes.

Five diets were tested : (1) Rowett Research Institute Stock Diet No. 86 (Howie, 1951). This is a cubed diet of a mixture of natural foods giving good growth and breeding performance. (2) Diet containing 30% casein. (3) Diet containing 20% casein. (4) Diet containing 10% casein. (5) Stock diet of bread soaked in milk daily, with a handful of oats weekly. The percentage composition of diets 1–4 is given in Table I.

*This work was done with a grant from the Medical Research Council.

TABLE I.—*Composition (g. per 100 g.) of Diets Tested*

Diet 1		Diets 2, 3, and 4			
			2	3	4
Wheat, whole ground	50	Casein (lactic, unextracted; Glaxo Labs.)	30	20	10
Barley, whole ground	25	Maize starch	61	71	81
White-fish meal	7				
Meat-and-bone meal	6				
Dried brewers' yeast	5				
Dried grass meal	5	Margarine*		3·0	
Cod-liver oil	1	Cod-liver oil		0·5	
Common salt (sodium chloride)	1	Salt mixture†		4·0	
		Dried yeast powder		1·5	
Total	100	Totals	100	100	100

* Ministry of Food special margarine containing 450–550 I.U. vitamin A per oz. and 90 I.U. vitamin D per oz.

† McCollum's salt mixture No. 185 with iron and potassium iodide (0·221 g. per kg.).

Experiment I

Young white Swiss female mice of about 6 weeks of age of the laboratory inbred strain, which had been bred and reared on diet 1, were allocated to one or other of diets 1 to 4, on which they were fed for three weeks before inoculation, and thereafter throughout the course of the experiment. Twenty mice were allocated to each group. The mice were kept in individual glass jars with sawdust litter, which was changed weekly, and with individual water-bottles. The "casein" diets were made up in bulk and kept in the refrigerator. Each mouse was given 5 g. of diet daily.

The inoculum was a 10% mixture in saline of blood at "acme" (see below) from four mice infected with *T. congolense* (Busimbi); 0.5 ml. was injected into each mouse subcutaneously. It may be explained that for over five years in this laboratory *T. congolense* (Busimbi strain) remained fully virulent for mice. The strain was maintained by passage to fresh mice whenever the number of trypanosomes in the blood of an infected mouse first became uncountable. This phase is known as "acme" and the strain derived thereby as an acme strain (Browning, Calver, and Adamson, 1948). The usual course of infection in a previously uninoculated animal is that small numbers of trypanosomes appear in the blood in three to six days, and increase rapidly until acme is reached (five to nine days). Parasitaemia persists up to a week thereafter (acute phase); then

TABLE II.—*Diet 1 (Stock Diet 86, Rowett Research Institute). Results of Infecting Mice with Trypanosomes*

No. of Days After Inoculation	Mouse Numbers																			
	101	102	103	104	105	106	107	108	109	110	111	112	113	114	115	116	117	118	119	120
4	−	−	−	+vl	+vl	−	−	−	−	−	−	−	+vl	−	−	−	−	+vl	−	−
5	+	−	−	++	+	+vl	+	+vl	+vl	−	−	+l	+	−	+vl	+vl	+	+	−	+
6	+++	+vl	+	+++	++	+	+++	+l	+++	+vl	+	+++	+++	+vl	++	++	+++	+++	+vl	+++
7	+++	+++	+++	+++	+++	+++	+++	+++	+++	+	+++	+++	+++	+++	+++	+++	+++	+++	+++	+++
9	+++	+++	+++	+++	+++	+++	+++	+++	+++	+++	+++	+++	+++	+++	+++	+++	+++	+++	+++	+l
10	+++	+++	+++	+++	+++	+++	+++	+++	Dead	+++	+++	+++	+++	+++	+++	+	+	+++	+++	−
11	+++	+++	+++	+++	+++	+++	+++	+++		+++	+++	+++	+++	+++	+++	−	−	+++	+++	−
12	+++	−	+++	+++	−	+++	+++	−		+++	+++	+++	+++	+	−	−	−	−	+++	−
13	Dead	−	+++	−	−	+++	+++	−		−	−	−	Dead	+++	−	−	−	−	−	−
14		−	+++	−	−	+	+++	−		−	−	−		+++	−	−	−	−	−	−
16		+	−	−	+++	−	+++	−		−	+	++		−	−	+	+	++	−	+l
17		+++	−	++	+++	−	+++	−		−	+++	+++		−	−	+++	+++	+++	+	+++
18		+++	+	+++	+++	−	−	+vl		+	+++	+++		+	+++	+++	+++	+++	++	+++
19		+++	+++	+++	+++	+	+	+++		+++	+++	+++		+++	+++	+++	+++	+++	+++	+++
20		+++	+++	+++	+++	+++	+++	+++		+++	+++	+++		+++	+++	+++	+++	+++	+++	+++
21		+++	Dead	+++	+++	+++	+++	+++		+++	+++	+++		+++	Dead	+++	+++	+++	+++	+++
23		+++		+++	+++	+++	+++	+++		+++	+++	+++		+++		+l	+++	+++	+++	+++
24		+++		+++	+++	+++	+++	+++		+++	+++	+++		+++		+++	+++	+++	+++	+++
25		+++		+++	+++	+++	+++	+++		+++	+++	+++		+++		+++	+++	+++	+++	+++
26		+++		+++	+++	+++	+++	+++		+++	+++	+++		+++		+++	+++	+++	+++	+
27		+++		+++	+++	+++	+++	+++		+++	+++	+++		+++		+++	+++	+++	+++	+
29		+++		+++	+++	+++	+++	+++		+++	+++	+++		+++		+++	+++	+++	+++	+++
30		+++		+++	+++	+++	+++	+++		+++	+++	+++		+++		+++	+	+++	+++	+++
31		+++		+	Dead	+++	+++	+++		+++	+++	+++		+++		+l	−	+++	+++	+++
32		+		+++		+++	+++	+++		+++	+++	+++		+++		+	−	+++	+	+++
33		++		+++		+++	+++	+++		+++	+++	+++		+++		+++	−	+++	+++	+++
34		+++		++		+++	+++	+++		+++	+++	+++		+++		+++	+vl	+++	+++	+++
35		+++		+++		+++	+++	+++		+++	+++	+++		+++		+++	+	+++	+++	+++
37		+++		+++		+++	+++	+++		+++	−	+++		+		+++	++	+++	+++	+++
38		+++		+++		+++	+++	+++		+++	−	+++		−		+++	+++	+++	+++	+++
40		+++		+++		+++	+++	+++		+++	+++	+++		+++		+++	+++	Dead	+++	+++

− to +++ = range of numbers of trypanosomes in blood (see text, p. 854). l = little; vl = very little.

trypanosomes disappear for about seven days, but reappear in the blood in large numbers and persist as a rule until the death of the animal (chronic phase). Death may take place in the acute phase or in the chronic phase. Animals may survive up to about four months after inoculation, but usually die much before that time.

In the present experiments the blood of each mouse was examined daily by pressing out a drop of blood between a cover-slip and slide and examining a wet film under the 1/6-in. (4-mm.) lens of the microscope. In recording the results of blood examination, the numbers of trypanosomes per microscope field were indicated in the following way :

–	=No trypanosomes seen after three minutes' search
+vl	=1 trypanosome in over 2 fields
+1	=1 trypanosome in 1-2 fields
+	=1-3 trypanosomes per field
++	=3-15 " " "
+++	=Over 15 " " "

Results

The results of daily blood examinations are given in Tables II to V. All the mice fed on the four different diets ate their food well and showed no signs of ill-health, having smooth sleek coats and gaining weight throughout the experiment until such time as those which died of the infections were actually dying. The weights of uninfected mice given diets 1 to 4 are shown in Table X.

Diet 1 (see Table II).—Trypanosomes first appeared in the blood (+vl, +1, +) of the animals of this dietary group four to six days after inoculation. They reached acme (+++) in six to nine days and persisted in numbers till 11 to 17 days after infection. Trypanosomes were absent from the blood for two to six days. Thereafter the infection, now chronic, continued with more or less constant numbers of trypanosomes in the blood until death or termination of

TABLE III.—*Diet 2 (30% Casein). Results of Infecting Mice with Trypanosomes*

No. of Days After Inoculation	Mouse Numbers																			
	121	122	123	124	125	126	127	128	129	130	131	132	133	134	135	136	137	138	139	140
4	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
5	–	–	+vl	–	–	–	–	+vl	–	–	–	–	–	–	+vl	–	–	–	–	–
6	–	–	+1	–	–	–	–	+	–	–	–	–	–	–	+1	+1	–	–	–	–
7	–	–	+	–	–	–	–	+1	–	–	–	–	+vl	–	–	++	–	–	–	–
9	–	–	+++	–	–	–	–	+	–	–	–	–	+++	–	–	+++	–	–	–	–
10	–	–	+++	–	–	–	–	–	–	–	–	+	+++	–	–	+++	–	–	–	–
11	–	–	+	–	–	–	–	–	–	–	–	–	+++	–	–	+++	–	–	–	–
12	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	Dead	–	–	–	–
13	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–		–	–	–	–
14	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–		–	–	–	–
16	–	–	++	–	+	++	–	–	++	–	–	–	+1	–	–		–	–	–	–
17	–	–	+++	–	+++	+++	–	–	+++	–	–	–	+++	–	–		–	–	–	–
18	–	–	+++	–	+++	+++	–	+	+++	+1	+	+	+++	–	–		–	–	–	–
19	–	–	+++	–	+++	+++	–	+++	+++	–	–	+1	+++	–	+vl		–	–	–	–
20	–	–	+++	–	+++	+++	Dead	+	Dead	–	–	–	+++	–	–		–	–	–	–
21	–	–	Dead	–	–	–	No	+++		–	–	–	+++	–	–		–	–	–	–
23	–	–		–	–	–	tryps	+++		–	–	–	Dead	–	–		–	–	–	–
24	–	–		–	–	–	in	+++		–	–	–		–	–		–	–	–	–
25	–	–		–	+1	+vl	heart	+++		+vl	–	+vl		–	–		–	–	–	–
26	–	–		–	++	++	blood	+++		+++	+1	+		–	–		–	+1	–	+
27	–	–		–	+++	+++		Dead		+++	+++	+++		–	–		–	–	–	–
29	–	–		–	+++	+++				+++	+++	Dead		–	–		–	–	–	–
30	–	–		–	Dead	+++				+++	+++			+vl	–		–	–	–	–
31	–	–		–		+++				+++	+++			–	–		–	+1	–	–
32	–	–		–		+++				Dead	+++			–	–		–	+	–	–
33	–	–		–		+++					+++			–	–		–	+++	–	–
34	–	–		–		+++					+++			–	+		–	+++	–	–
35	–	–		–		+++					+++			–	+++		–	+++	–	–
37	–	–		–		Dead					+++			–	+++		–	+++	–	+1
38	–	–		–							+++			+	+++		–	+++	–	+++
40	–	–		–							Dead			+++	+++		–	+++	–	+++

TABLE IV.—*Diet 3 (20% Casein). Results of Infecting Mice with Trypanosomes*

No. of Days After Inoculation	Mouse Numbers																			
	141	142	143	144	145	146	147	148	149	150	151	152	153	154	155	156	157	158	159	160
4	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	Dead	–	–
5	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–		–	–
6	–	–	–	–	–	–	–	–	+	–	–	+	–	–	–	–	–		–	–
7	+	–	–	–	–	–	–	–	+++	–	–	+++	–	–	–	–	–		–	–
9	–	–	–	–	–	+1	–	–	Dead	–	–	+++	–	+1	–	–	–		–	–
10	–	–	–	–	–	–	–	–		–	–	–	–	++	–	–	–		–	–
11	–	–	–	–	–	–	–	–		–	–	–	–	+++	–	–	–		–	–
12	–	–	–	–	–	–	–	–		–	–	–	–	+++	–	–	–		–	–
13	–	–	–	–	–	–	–	–		–	–	–	–	+++	–	–	–		–	–
14	–	–	–	–	–	–	–	–		–	–	–	–	Dead	–	–	–		–	–
16	–	–	–	–	–	–	–	–		–	–	–	–		–	–	–		+	–
17	+1	–	–	–	–	–	–	+		–	+	+	–		–	–	–		+	–
18	+++	–	–	–	–	+vl	–	–		–	+++	++	–		–	–	–		+++	–
19	+++	–	–	–	–	++	+vl	–		–	+++	++	–		–	–	–		+++	+vl
20	–	–	–	–	–	+++	–	–		–	–	+++	–		–	–	–		+++	–
21	–	–	–	–	–	+++	–	–		–	–	+++	–		–	–	–		+++	–
23	–	–	–	+1	–	+++	–	+1		–	–	+++	–		–	–	–		Dead	–
24	–	–	–	+++	–	+++	–	–		–	–	+++	–		–	–	–			–
25	–	–	–	+++	–	+++	–	–		–	+vl	+++	–		–	–	–			–
26	+vl	–	–	+++	–	+++	–	–		–	++	+++	–		–	–	–			–
27	+1	–	–	+++	–	+++	–	–		–	+++	+++	–		–	–	–			–
29	+	–	–	+++	–	+++	–	+++		–	+++	Dead	–		–	–	–			–
30	++	–	+vl	Dead	–	Dead	–	+++		–	+++		–		–	–	–			–
31	+1	–	–		–		–	Dead		–	–		–		–	–	–			–
32	++	+vl	–		–		–			–	–		–		–	–	–			–
33	+++	–	–		–		–			–	–		–		–	–	–			–
34	+	–	–		–		–			–	–		–		–	–	–			–
35	–	–	–		–		–			–	–		–		–	–	–			–
37	–	–	–		–		–			–	–		–		–	–	–			–
38	–	–	–		–		–			–	–		–		–	–	–			–
40	–	–	–		–		–			–	–		–		–	–	–			–

– to +++=range of numbers of trypanosomes in blood (see text, above). l=little; vl=very little.

TABLE V.—*Diet 4 (10% Casein). Results of Infecting Mice with Trypanosomes*

No. of Days After Inoculation	Mouse Numbers																			
	161	162	163	164	165	166	167	168	169	170	171	172	173	174	175	176	177	178	179	180
4	−	−	−	−	−	−	+l	−	−	−	−	−	−	−	−	−	−	−	−	−
5	−	−	−	−	−	−	++	−	−	−	−	−	−	−	−	−	−	−	−	−
6	−	−	−	−	−	−	+++	−	−	−	−	−	−	−	−	−	−	−	−	−
7	+	+l	−	+l	−	+l	+++	−	+l	+	−	−	−	+	−	−	−	+l	−	−
9	−	−	−	+++	−	−	Dead	−	−	+++	+vl	−	−	+++	−	−	−	−	−	−
10	−	−	−	Dead	−	−		−	−	+++	−	−	−	+++	−	−	−	−	−	−
11	−	−	−		−	−		−	−	+++	−	−	−	+++	−	−	−	−	−	−
12	−	−	−		−	−		−	−	Dead	−	−	−	+++	−	−	−	−	−	−
13	−	−	−		−	−		−	−		−	−	−	+++	−	−	−	−	−	−
14	−	−	−		−	−		−	−		−	−	−	Dead	−	−	−	−	−	−
16	+vl	+vl	−		−	−		−	−		−	−	−		−	−	−	+	++	−
17	+l	++	−		+l	−		+l	−		−	−	+		−	−	−	+++	+++	−
18	−	+++	−		−	−		+	−		−	−	++		−	−	−	+++	+++	−
19	−	+++	−		−	−		++	−		−	−	+++		+vl	−	−	+++	+++	−
20	−	+++	−		−	−		++	−		−	−	+++		++	−	−	+++	+++	−
21	−	+++	−		−	−		+++	−		−	−	+++		+++	−	−	+++	+++	−
23	−	Dead	−		−	−		+++	−		+l	−	+++		Dead	−	−	+++	+++	−
24	−		−		−	−		+++	−		−	−	+++			−	−	+++	Dead	−
25	+vl		−		+vl	−		+++	−		−	−	Dead			−	−	+++		−
26	++		−		++	+vl		+++	−		−	−				+vl	−	Dead		−
27	+++		−		+++	+		+++	−		−	−				−	−			−
29	+++		−		+++	+		+++	+vl		−	−				−	−			−
30	+++		−		Dead	+		−	++		−	−				−	−			−
31	+++		−			+++		+++	+++		+vl	−				−	−			−
32	+++		−			+++		Dead	+++		+++	−				−	−			−
33	+++		−			+++			+++		+++	−				−	−			−
34	Dead		−			+++			+++		+++	−				−	−			−
35			−			+++			+		Dead	−				−	−			−
37			−			+++			+			−				+vl	−			−
38			−			Dead			+			−				+	−			−
40			−						+			−				+++	−			−

− to +++=range of numbers of trypanosomes in blood (see text, p. 85). l=little; vl=very little.

the experiment. All animals were successfully infected. During the 40-day period of observation three mice died at the acute stage and four at the chronic stage of the infection—that is, 7 out of 20 died.

Diet 2 (see Table III).—Trypanosomes first appeared in the blood of 5 of the 20 mice in five to seven days, and of these only three showed the normal course of increase to +++ with relapse or death at the acute stage. Of the remaining 15 mice, six did not at any time show evidence of infection in the blood, and in the other nine the first demonstrated appearance of trypanosomes was from 10 to 26 days after inoculation. In 7 of the 20 mice, trypanosomes in very small numbers appeared in the blood for a very short time, sometimes on more than one occasion, before they reached +++, after which the course of persistent chronic infection always followed till death. During the 40 days 10 of the 20 mice died of infection—only one of these died at the acute stage—and one mouse (No. 127) died of intercurrent infection. The five mice in which trypanosomes had not been demonstrated were reinoculated from a fresh mouse of the acme passage strain, and their blood was examined for a further 15 days, during which time no sign of infection was observed.

Diet 3 (see Table IV).—One mouse (No. 158) died of intercurrent infection at the beginning of the experiment. Five of the 19 mice showed trypanosomes in the blood from six to nine days after inoculation; of these only two increased to +++ at this stage. Of the remaining mice, eight showed trypanosomes in 16 to 32 days, but only four of the eight ever showed a +++ infection or reached the chronic phase—that is, four mice showed trypanosomes but never at the level of +++. In 6 of the 19 mice, trypanosomes were not demonstrated. These animals also were reinoculated as described before, but no resulting infection was seen. Seven of the 19 mice died during the 40 days, two at the acute stage.

Diet 4 (see Table V).—Ten of 20 mice showed trypanosomes in the blood in four to nine days, four reached the +++ level and died at the acute stage. Six further mice showed trypanosomes after 16 to 26 days, all of these mice eventually showing a +++ infection which became chronic. In four mice trypanosomes were not demonstrated, and these were reinoculated as before. Trypanosomes were not seen after reinoculation. In this group there were 14 deaths—four at the acute and 10 at the chronic stage of infection.

The results of experiment I are summarized in Table VI.

TABLE VI.—*Summary of Data from Tables II-V*

Dietary Group (20 Mice in Each Group)	No. of Mice Showing Trypanosomes in Blood Within 9 Days	No. of Mice Reaching + + + Within 9 Days	No. of Deaths			No. of Mice in Which Trypanosomes Were Not Demonstrated in the Blood	No. of Mice in Which Trypanosomes were Seen but did not Reach +++ Level
			At "Acute" Stage	At "Chronic" Stage (Up to 40 Days)	Total		
1	20	20	3	4	7	0	0
2	5	3	1	9	10	5	0
3*	5	2	2	5	7	6	4
4	10	4	4	10	14	4	0

* One of the mice given diet 3 died of intercurrent infection at the start of the experiment.

Experiment II

This experiment was done with diet 1, diet 3, and diet 5, which consisted of bread, milk, and oats. Diet 5 was that used for trypanosome-infected mice in this department for many years.

Details of inoculation and blood examination were similar to those in Experiment I. Male mice of the laboratory inbred strain 3–4 months of age were allocated to the three diets, on which they were fed for a period of two weeks before inoculation with *T. congolense* (Busimbi). The period of observation after inoculation was only 23 days in this experiment.

Results

Diet 1 (see Table VII).—Trypanosomes first appeared in the blood of infected animals in four to eight days after inoculation. Acme was reached at 6 to 10 days. The trypanosomes disappeared from the blood on the eleventh to the fifteenth day after inoculation and reappeared in four to nine days after disappearance. All the animals became infected. One mouse died at the acute stage of the infection, but 9 of 10 mice survived to the chronic stage, at which all remained well till the end of the experiment.

Diet 3 (see Table VIII).—In this group only 2 of 10 mice showed trypanosomes in blood films in six to seven days. Of these two, one had demonstrable numbers of

trypanosomes on only one day of examination, while the other, in which trypanosomes persisted over three days, did not reach acme.

Diet 5 (see Table IX).—Trypanosomes first appeared in the blood of animals in this group five to seven days after infection. Acme was reached at seven to eight days. All the animals became infected. Trypanosomes persisted in large numbers in the blood for as long as 18 days after inoculation, when they disappeared for up to five days. Four mice died at the acute stage, but there were no deaths at the chronic stage in this period of observation.

Discussion

In Experiment I all the animals in the group fed on diet 1 followed the course of infection normally observed in this laboratory with *T. congolense* (Busimbi) (see Table II). The results of the infection in mice given diets 2, 3, and 4, on the other hand, repeated the unexpected result referred to in the introduction of many failures to infect in the normal manner with this strain. Only 5 out of 20 mice on each of diets 2 and 3 and 10 of 20 mice on diet 4 showed trypanosomes in their blood in anything approaching the normal times; and of these numbers only three animals on diet 2, two on diet 3, and four on diet 4 showed the normal acme phase (Table VI). The remaining mice relapsed before the acute stage of infection had been reached, and did not show the usual chronic phase which normally persists for variable but substantial periods of time. In the other animals on diets 2, 3, and 4 trypanosomes were not demonstrated in the blood for up to as long as 26, 32, and 26 days (Tables III, IV, and V), and indeed were not demon-

TABLE VII.—*Diet 1 (Stock Diet 86). Experiment II: Results of Infecting Mice with Trypanosomes*

No. of Days After Inoculation	Mouse Numbers									
	201	202	203	204	205	206	207	208	209	210
4	–	–	–	–	–	–	–	–	+vl	–
5	+l	–	+vl	–	–	–	–	+vl	+vl	+vl
6	+	+vl	++	+vl	+vl	+vl	–	+++	+l	+
7	+++	++	+++	++	+	+++	–	+++	+++	+++
8	+++	+++	+++	+++	+++	+++	+vl	+++	+++	+++
9	+++	+++	+++	+++	+++	+++	++	+++	+++	+++
11	+++	+++	–	+++	Dead	+++	+++	+++	–	+++
12	+++	+++	–	+++		–	+	–	–	+++
13	–	+++	–	–		–	–	–	–	–
14	–	+	+	–		–	–	–	–	–
16	–	–	+++	–		–	+	–	++	–
18	+l	–	+++	+l		–	+++	++	+++	++
19	+++	–	+++	+++		–	+++	+++	+++	+++
20	+++	+	+++	+++		+	+++	+++	+++	+++
21	+++	++	+++	+++		+++	+++	+++	+++	+++
22	+++	+++	+++	+++		+++	+++	+++	+++	+++
23	+++	+++	+++	+++		+++	+++	+++	+++	+++

TABLE VIII.—*Diet 3 (20% Casein). Experiment II: Results of Infecting Mice with Trypanosomes*

No. of Days After Inoculation	Mouse Numbers									
	211	212	213	214	215	216	217	218	219	220
4	–	–	–	–	–	–	–	–	–	–
5	–	–	–	–	–	–	–	–	–	–
6	–	–	–	–	+vl	–	–	–	–	–
7	+vl	–	–	–	+l	–	–	–	–	–
8	–	–	–	–	+	–	–	–	–	–
9	–	–	–	–	–	–	–	–	–	–
11	–	–	–	–	–	–	–	–	–	–
12	–	–	–	–	–	–	–	–	–	–
13	–	–	–	–	–	–	–	–	–	–
14	–	–	–	–	–	–	–	–	–	–
16	–	–	–	–	–	–	–	–	–	–
18	–	–	–	–	–	–	–	–	–	–
19	–	–	–	–	–	–	–	–	–	–
20	–	–	–	–	–	–	–	–	–	–
21	–	–	–	–	–	–	–	–	–	–
22	–	–	–	–	–	–	–	–	–	–
23	–	–	–	–	–	–	–	–	–	–

TABLE IX.—*Diet 5 (Natural Diet of Bread, Milk, and Oats).Experiment II: Results of Infecting Mice with Trypanosomes*

No. of Days After Inoculation	Mouse Numbers									
	221	222	223	224	225	226	227	228	229	230
4	–	–	–	–	–	–	–	–	–	–
5	–	–	–	–	–	–	+	–	+vl	–
6	–	+vl	–	+	++	–	+vl	++	++	++
7	+	+	+l	+++	+++	+	++	+++	+++	+++
8	+++	+++	+++	+++	+++	+++	+++	+++	+++	+++
9	+++	+++	+++	+++	+++	+++	+++	+++	+++	+++
11	+++	Dead	+++	Dead	–	+++	+++	+++	Dead	+++
12	+++		+++		–	+++	+++	+++		+++
13	+++		+++		–	–	+++	–		+++
14	+++		+		–	–	+++	–		+++
16	Dead		–		+	+	–	+		+++
18			+++		+++	+++	+++	+++		+++
19			+++		+++	+++	+++	+++		+vl
20			+++		+++	+++	+++	+++		–
21			+++		+++	+++	+++	+++		–
22			+++		+++	+++	+++	+++		+
23			+++		+++	+++	+++	+++		+++

– to +++=range of numbers of trypanosomes in blood (see text, p. 854). l=little; vl=very little.

strated at all in five, six, and four animals of the respective dietary groups 2, 3, and 4, even after reinoculation with the blood of a fresh "acme" passage mouse. In mice given diet 2, 3, or 4 infection followed a much milder course than in those given diet 1. This was true in all mice not reaching or dying at the acute stage, as judged by the total numbers of trypanosomes in the blood of the animals over the period of the experiment. When the persistent chronic stage was reached, however, death was often rapid (cf. Table II and Tables III, IV, and V).

The results of Experiment I are supported by those of Experiment II. The mice fed on the casein diet 3 showed only the slightest evidence of blood-stream invasion by parasites during the 23-day period of observation. The infection followed an acute course in both the natural diets, Nos. 1 and 5, and this course was that normally recorded in this laboratory over many years.

Maegraith *et al.* (1952) concluded that milk probably contains something that can inhibit or restrict the development of the asexual phase of blood-transmitted *Plasmodium berghei* in the rat and the mouse. Another possibility is that milk may be deficient in certain nutrients required by the parasite (*British Medical Journal*, 1952). The second explanation would fit the results observed in the present experiments with *T. congolense* in mice given diets with milk casein as the source of protein. It is clear that parasitaemia developed much more rapidly and attained a higher level in mice given diets 1 and 5, which were nutritionally more complex than the casein diets. Moreover, since diet 5 also contained milk, inhibition of parasite growth from the absence of undefined nutrients rather than from an anti-parasitic effect of milk might appear to be the more tenable explanation. It may be of importance to note that the mice made good gains of weight on all diets, especially on diet 4, which contained a high proportion of carbohydrate (Table X).

TABLE X.—*Weekly Mean Weights (g.) of Uninfected Mice Given Experimental Diets 1–4*

Diet No.*	Age of Mice (Weeks)								
	4	5	6	7	8	9	10	12	17
1	19	24	29	30	31	32	34	36	39
2	17	25	30	30	32	32	33	35	35
3	20	24	30	33	33	35	37	37	37
4	18	25	32	34	36	37	39	41	44

* Each dietary group comprised 4 males and 4 females; litter mates were evenly distributed.

Summary

Mice fed on five different diets were experimentally infected with *T. congolense*.

Parasitaemia with acute followed by chronic infection developed as expected in mice fed on two of the diets, which were diets of the type normally employed in laboratories for rearing and maintenance of mice.

In mice fed on three of the diets in which milk casein was the source of protein the parasitaemia was either delayed or prevented.

Since one of the two "normal" diets contained milk but permitted normal development of the infection it might appear that failure of the incomplete diets to nourish the parasite is the explanation of the observed results, rather than that milk itself contains an anti-parasitic agent.

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HISTOPLASMOSIS IN GREAT BRITAIN

DESCRIPTION OF A SECOND CASE OF DISSEMINATED HISTOPLASMOSIS; TREATMENT BY ETHYL VANILLATE

BY

S. LOCKET, M.B., B.S., M.R.C.P.
Physician-in-Charge, Medical Unit

E. A. ATKINSON, M.B., B.S., M.R.C.S., L.R.C.P.
Pathologist-in-Charge, Department of Pathology

AND

W. S. M. GRIEVE, M.Sc., Ph.D., F.R.I.C.
Biochemist

With the technical assistance of E. BRIDSON, F.I.M.L.T.

Oldchurch Hospital, Romford, Essex

Work in the past few years on human infections by the *Histoplasma capsulatum* has emphasized certain features. Generalized dissemination (disseminated histoplasmosis) is common and usually fatal. Infection confined to the skin is frequent, though, unlike cutaneous blastomycosis, the skin lesion is usually within a short while followed by manifestations of dissemination (Curtis and Grekin, 1947) and is therefore often fatal. Infection can be confined to a single organ or viscus—usually the lung—with at no time evidence of dissemination and often no past history of even mild illness. These cases are identified usually after recovery by a positive intradermal histoplasmin test or by complement fixation.

Only one other case of disseminated histoplasmosis has been reported in Great Britain, and this occurred in 1940 (Derry, Card, Wilson, and Duncan, 1942). A regular soldier aged 30 developed his illness in France in 1940 and the diagnosis was eventually made at necropsy in Great Britain. He had been resident in India in 1932–6 and in the Sudan in 1936–7; this gives a possible incubation period of three years at least (assuming the disease is not indigenous to France).

In the second case, which we report below, the patient had not been out of England for seven years. Symptoms first appeared about a year before admission to hospital, and we must therefore assume either that the disease has an incubation period of at least six years or that he contracted his illness in England. We regard the first of these suggestions as the more probable.

Arblaster (1950) and Crofton (1950) report three cases of pulmonary calcification in England in which the diagnosis of "healed" histoplasmosis was suggested because of a positive histoplasmin intradermal test. All three patients had been resident in the U.S.A. or Canada. Duncan (1945, 1947) isolated an atypical organism from a skin lesion in a man from West Africa.

In 1951 Limerick described the development of a retrocaecal abscess occurring after appendicectomy in 1947 in a man aged 35. The abscess cleared up in three weeks with drainage and was said to be due to *H. capsulatum*. No further details are given.

The cases of Duncan, Arblaster, and Limerick, however, do not conform in many ways to the usually accepted criteria and must be regarded as very doubtful. Hutchison (1952) reported a localized laryngeal lesion due to *H. capsulatum* in a fit man of 72 who had lived

in Hyderabad for 30 years and had spent some time in Malaya. He had, however, been resident in Great Britain since 1934 (six years in Sussex and eight years in Dumfriesshire until the diagnosis in 1948). He was fit and well in 1951.

These cases are particularly interesting on two counts: (1) the three patients, not from the American continent, had all been to India; and (2) in view of the prolonged incubation period it is likely that further cases may now be expected to occur in this country in soldiers who had been resident in endemic areas during the war.

Case History

The patient, a man aged 32, had been in H.M. Forces for six years and was discharged in England in 1945. He had spent most of his time at Alexandria and Port Said, but had visited Durban for three weeks, had spent three weeks in Tobruk, and had stayed at Bombay for two months. He visited numerous places in the Near East, including Cyprus, Aden, and Malta, but never went ashore.

Fig. 1.—Granulomatous ulcer inside right cheek.

He began to complain of peptic-ulcer-like abdominal pain at the end of 1951. On October 14, 1952, he first attended this hospital, and he was seen by one of us (S. L.) on December 17. On examination he was pyrexial, with obvious loss of weight, a large spleen, a large liver, a few small axillary glands, pallor, marked tenderness over the sternum, and two large ulcers in the mouth (see Fig. 1). Examination of the central nervous system was normal; the blood pressure was 125/75 mm. Hg. The ulcers (about 2 cm. in diameter) were situated on the hard palate and the inside of the right cheek. They were granulomatous in appearance, with a raised rolled edge. The patient was admitted to the medical wards.

On December 19 a blood count showed: haemoglobin, 58%; red cells 3,300,000; white cells, 3,000 (72% polymorphs, 1% metamyelocytes, 1% eosinophils, 11% lymphocytes, 8% monocytes, 7% stab cells). A marrow puncture was normal and did not favour the diagnosis of leukaemia. W.R. and Kahn test were negative. It was felt that the only alternative diagnosis now possible was that of disseminated histoplasmosis.

Penicillin in large doses and the local application of an antibiotic to the ulcers was ineffective, and was discontinued after ten days. An *x*-ray film of the chest on December 29 showed the presence of a small opacity in the right costophrenic angle. Intravenous hydroxy-stilbamidine for ten days was ineffective. On January 7, 1953, his haemoglobin was 54% and the blood count was unchanged. Repeated estimations of plasma proteins, serum bilirubin, thymol turbidity, and cephalin-cholesterol flocculation were unchanged and normal. Blood urea was 26 mg. per 100 ml. A histoplasmin intradermal test was negative (no wheal or erythema). An electrocardiogram on January 14 was within normal limits.

An *x*-ray film of the chest on January 21 showed extensive spread of the pulmonary lesion (see Fig. 2). On the 24th small ulcers of the skin were appearing and doses of sulphadiazine were started with no improvement. On the 29th ethyl vanillate was given.

On February 1 he was having great difficulty in keeping down the capsules of ethyl vanillate, and from February 5 it was given in an emulsion. The emulsion was made up to contain 12.5 g. of ethyl vanillate in 200 ml., and this was dripped into the stomach through a Ryle's tube, the gastric drip lasting for 50 minutes and being given every six hours.

On February 9 he said he felt very much better, but was even more wasted. His blood pressure was 115/70 mm. Hg. There had been no improvement in any of his physical signs, and his clinical condition had grossly deteriorated. He died suddenly on the evening of that day.

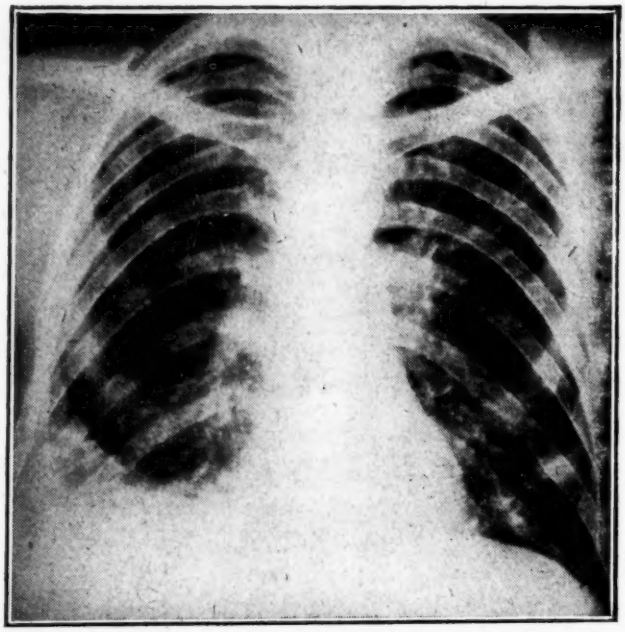

Fig. 2.—Chest *x*-ray film two weeks before death, showing extensive bilateral lung involvement.

Post-mortem Findings

The liver weighed 1,552 g., and a large number of pale slightly raised nodules up to 1.5 cm. in diameter could be seen through the capsule. Enlargement of lymph nodes was general, but was especially marked in the abdomen. The suprarenals showed enormous enlargement, presenting as yellowish rounded masses 6 by 6 by 4 cm., on each side deforming the upper pole of each kidney. On section the suprarenals had a cream-coloured honeycombed appearance with some liquefaction. The kidneys and bowel showed no detectable lesions. The spleen weighed 430 g., with a splenunculus the size of a walnut. The lungs each weighed just over 600 g., all lobes showing very numerous white solid areas 1–5 mm. in diameter, sometimes coalescing into larger masses with central necrosis.

Histological evidence of histoplasmosis was found in sections from the liver, spleen, lymph nodes, lungs, bone marrow, thyroid, and suprarenals. Very scanty parasites were detected in sections of skin lesions. In each case distension of reticulo-endothelial cells by large numbers of capsulated yeasts was evident (Fig. 3). Goodpasture's stain gave clearer definition of the parasites than Schiff's periodic acid or phloxine tartrazine. In the suprarenals there was extensive involvement of the cortex as well as the medulla, with a very small outer fringe of unaffected cells.

H. capsulatum was cultivated from the spleen, suprarenals, liver, lung, and bone marrow.

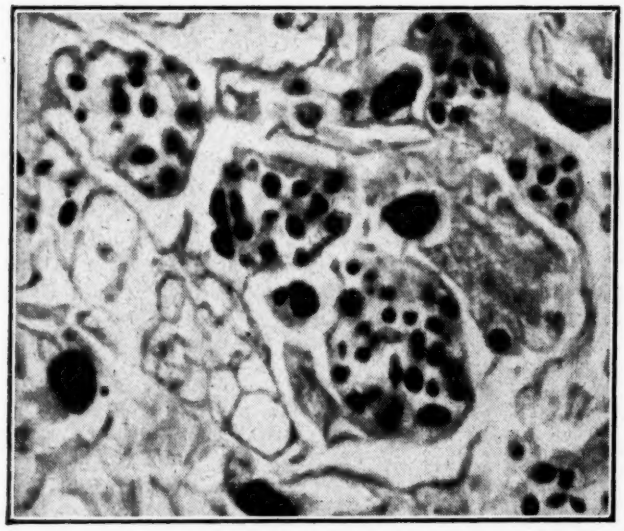

FIG. 3.—Section of lymph node showing endothelial cells engorged with the parasites.

Bacteriology

Examination of the first biopsy (December 29) was so suggestive of histoplasmosis that a second biopsy was carried out (January 21) in order to isolate the parasite. An impression smear (January 21) showed an abundance of oval capsulated yeasts (Fig. 4), and cultures were made on Sabouraud's and glucose agar (*p*H 7.4) at room temperature and at 37° C. On 1% glucose agar after seven days at room temperature there appeared a profuse growth of small fluffy white colonies adherent to the medium. A few days later typical tuberculate chlamydospores of *H. capsulatum* were found in the centre of the colonies. No growth occurred on Sabouraud medium, and it appears that, from subculturing, this strain grows very slowly at a low *p*H. On glucose agar, growth was best at room temperature at a *p*H approaching neutrality. The colonies become brownish, with the appearance of chlamydospores. The chlamydospores (Fig. 5) are of two types—a large tuberculated thick-walled structure 14–16 μ in diameter and a small smooth pear-shaped form 4–5 μ in diameter. From the mycelial growth cultures were made on sealed blood-agar slants at 37° C., and after a fortnight smooth creamy colonies of the yeast form were produced. The yeast cells were of varying shape, about 2–3 μ in diameter, and about half appeared oval with pointed ends. The yeast form did not ferment glucose, maltose, saccharose, or lactose after 14 days' incubation at 37° C.

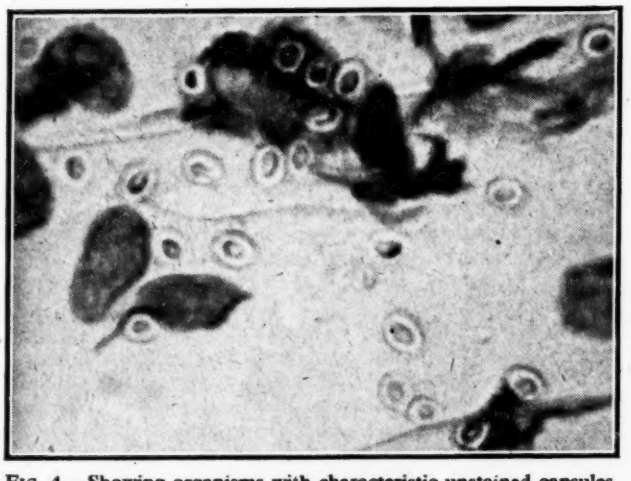

FIG. 4.—Showing organisms with characteristic unstained capsules obtained by direct contact smear from biopsy of cheek ulcer. (×2,000.)

With the use of the slide-cell technique and 1% glucose agar the control cell showed a good growth in six days, but no growth occurred in concentrations of 4.2 mg. of ethyl vanillate or greater. Similar tests carried out with "cuprimyl" (tetradiethylamine salt of cupric-*bis*-8-hydroxyquinoline 5:7 disulphonic acid) showed no inhibition in a concentration of 10 mg. per 100 ml.

Discussion

In this case there did not appear to be any evidence of adrenal failure, though a close watch was kept for this both biochemically and clinically; nevertheless the sudden death was very suggestive of this.

The diagnosis of the disseminated disease can in most cases be suspected on the clinical picture: irregular fever, ulceration of the mouth and palate of a peculiarly granulomatous type (Fig. 1), ulceration of the skin, enlargement of the liver and spleen, lymph-node enlargement, developing pulmonary lesions which may show a localized consolidation with or without cavitation (Fig. 2), or, more commonly, generalized miliary opacities in the lungs. Anorexia and much loss of weight, to eventual gross cachexia, are the rule. Abdominal pain and discomfort is common, and intestinal ulceration with diarrhoea not infrequent. The diagnosis can be confirmed, however, only by identification of the organism in the tissues (Fig. 3) and positive culture.

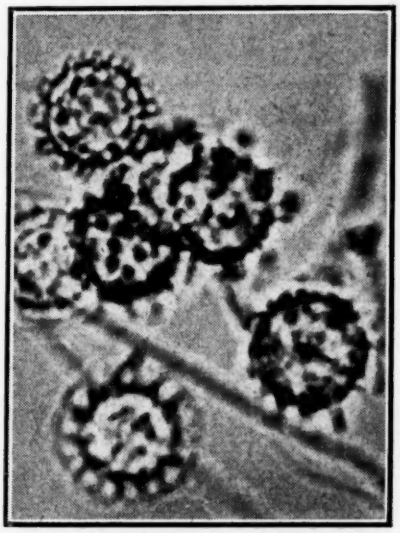

FIG. 5.—Characteristic "cog-wheel" appearance of chlamydospores seen on culture of *H. capsulatum*. (×2,000.)

Treatment

Clinical experience has shown the complete failure of promanide (promin in U.S.A.), "actidione" (an antibiotic from *Streptomyces griseus*), penicillin, streptomycin, aureomycin, chloramphenicol, sulphonamides, iodides, arsphenamine, mercurials, stilbamidine, and antimonials to affect the course of the disease and its fatal termination, and to this list we would add hydroxy-stilbamidine. Two cases have been reported in which pulmonary resection has apparently resulted in survival for two years and for six months respectively (Hodgson, Weed, and Clagett, 1950).

Ethyl vanillate, obtained by oxidizing lignin in the spent liquor from the sulphur pulp manufacturing process, has been used in disseminated histoplasmosis (Christie, Middleton, Peterson, and McVickar, 1951; Ellis, Scott, and Miller, 1952), being at the moment the drug of choice in treating histoplasmosis and coccidioidomycosis. In concentrations of 10 to 30 mg. per 100 ml., it has a marked inhibiting effect on *Blastomyces dermatitidis, Coccidioides immitis,* and *H. capsulatum* (information provided by Messrs. Squibb). However, a difference in susceptibility to ethyl vanillate has been demonstrated for different strains of *H. capsulatum* which may be of significance in treatment. It is, however, rather surprising that the organism in our case did not grow in ethyl vanillate at a concentration of 4.2 mg. per 100 ml., yet, in spite of blood levels well above this for 10 days (see Table), the disease here showed no signs of regression.

Ethyl Vanillate Levels in Blood and Urine (24-hour Collections)

Day	Plasma Vanillic Acid (Corrected for Normal Phenolic Substances in Blood) (mg./100 ml.)	Urine Vanillic Acid (Calculated as Vanillic and Phenolic Substances Excreted (g.))
28/1/53		1·70
29/1/53		0·88
30/1/53	6·8	3·70
31/1/53		16·50
1/2/53		14·40
2/2/53	3·3	5·75
3/2/53	18·6	26·10
4/2/53	15·8	
5/2/53	20·5	11·40
6/2/53		4·83
7/2/53		6·42
9/2/53	21·5	

Blood levels of over 30 mg. per 100 ml. are apt to cause toxic phenomena (coma with or without convulsions), and if the level exceeds 50 mg. the toxic phenomena can be lethal (Christie *et al.*, 1951). To maintain this blood level in an adult requires about 40 to 50 g. of ethyl vanillate daily by mouth, and in a child 0.75 to 2.5 g. per kg. of body weight a day. More recently, Christie (1952) said that ethyl vanillate was the only therapeutic agent of any value in histoplasmosis, though it was not satisfactory.

The patient here described received 45 g. daily from February 2, but part of this was vomited. From February 5 he received and retained 50 g. daily.

Summary

After a brief review of the cases of histoplasma infection previously published in Great Britain the second case of disseminated histoplasmosis to be reported is described. This case was fatal in spite of treatment with 45–50 g. of ethyl vanillate daily. Some information is included on culture of the organism, as in this case it showed certain cultural peculiarities.

We are grateful for the help we received from many sources in the treatment of this case. We would particularly like to thank Dr. A. S. Osborne, of the American Embassy, Dr. W. P. Kennedy, of the Ministry of Health, and Mr. C. B. Sitterson, jun., of the Institute of Paper Chemistry, Wisconsin, U.S.A., for their advice; Dr. Harvey Blank and Messrs. E. R. Squibb and Sons, New York, U.S.A., for the prompt dispatch to us of ethyl vanillate and appropriate information adequate for the treatment of this case; Dr. R. Forgan, Dr. John Harper, and Messrs. May and Baker, of Dagenham, for their advice and for supplying us with the hydroxy-stilbamidine and "cuprimyl" used by us; Mrs. E. D. Price for her patience and careful secretarial work; and Mr. E. Sheppard for his most excellent photography. Finally, we are most grateful to the nursing staff for the great care with which treatment was carried out, and to Dr. G. H. Cranswick and Dr. P. G. Swann for their many helpful suggestions.

Since this paper was completed we understand that a case of disseminated histoplasmosis in a young woman was transferred to this country from South Africa, where the diagnosis was made. Unfortunately the patient died two days after arrival.

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THE MECHANISM OF SYNCOPE AFTER COUGHING

BY

E. P. SHARPEY-SCHAFER, F.R.C.P.
Professor of Medicine, London University
(From the Department of Medicine, St. Thomas's Hospital)

Syncope from coughing has been considered a rare condition; thus Whitty (1943) and Baker (1949) could collect only about 100 cases from the literature. Charcot (1876) thought it was a form of epilepsy, and by coining the term "laryngeal vertigo" made it the province of neurology and throat surgery. McCann *et al.* (1949) obtained some continuous records during coughing in two subjects with the syndrome. They believed that syncope was not primarily epileptic but circulatory in origin, and that pulmonary arterial vasoconstriction or pathologically sensitive "vascular reflexes" might be responsible. Correspondence in the *British Medical Journal* resulted in no factual data, but suggested that the condition might be commoner than supposed.

Material and Methods

The patients were 27 middle-aged or elderly males who gave a history of syncopal attacks on coughing. In many it was found possible to provoke an attack by asking the patient to give a few strong coughs. Inhaling cigarette smoke or irritation of the nose was used in a few. Pressures were recorded continuously by capacitance manometers (Hansen, 1949): intrathoracic by means of a water-filled oesophageal tube (Dornhorst and Leathart, 1952); intracardiac by catheter; and intra-arterial and venous by needles or fine catheters. Forearm flow was measured with a plethysmograph and recorded on the same photographic paper (Davies, 1953).

Results

The data obtained in the 27 subjects with syncope were compared with similar measurements in about 40 other subjects of all ages and both sexes. There was no qualitative difference in the effects of coughing in the two groups. A single "ahem" produced an intrathoracic pressure rise of 20 to 30 mm. Hg, ordinary coughing in both groups 50 to 100 mm. Hg, and coughing leading to syncope 150 to 300 mm. Hg. The circulatory effects were proportional to the violence and duration of the coughing attacks. All subjects performed the Valsalva manœuvre. There was no difference in circulatory effects after the Valsalva manœuvre between those with cough syncope and those without cough syncope.

Continuous records of syncopal attacks were obtained on fifteen occasions, and many minor attacks causing dizziness or "blackout" were also recorded. The subjects coughed in two ways, continuous or intermittent, with different circulatory effects. Both, when violent, resulted in syncope.

Continuous Coughing

The subjects coughed continuously in prolonged paroxysms lasting many seconds without drawing breath. Often the intrathoracic pressure reached 300 mm. Hg and was maintained at this level until a breath was taken or the paroxysm ended. The circulatory effects were essentially similar to those of the Valsalva manœuvre, but more pronounced owing to the greater rise of intrathoracic pressure. When asked to perform their maximum Valsalva manœuvre it was found that few could reach and maintain an intrathoracic pressure of 100 mm. Hg. This conspicuous

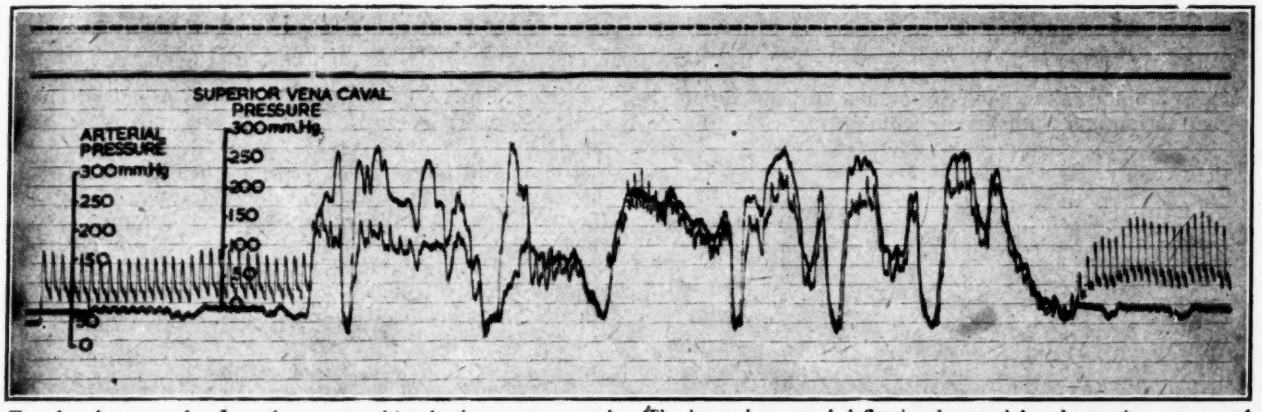

FIG. 1.—An example of continuous coughing lasting many seconds. The large downward deflection in arterial and superior vena caval pressures during the attack are due to occasional inspirations. Consciousness was lost at the end of the attack, when arterial pressure was lowest. Time marker in seconds in this and subsequent records.

difference between coughing and the Valsalva manœuvre may result from the greater number of muscles and the more powerful intermittent muscular effort employed in coughing.

An example of syncope from continuous coughing is shown in Fig. 1. Unconsciousness resulted when arterial pressure was lowest at the end of the attack. As in other forms of syncope from acute arterial hypotension (Barcroft *et al.*, 1944; Brigden *et al.*, 1950; Howard *et al.*, 1951; Howarth and Lowe, 1953) the critical level for loss of consciousness was about 50 mm. Hg. Twitching movements and convulsions were common during syncope.

Two subjects were observed to become unconscious, as judged by rolling up of the eyes and twitching movements, during coughing, while intrathoracic and arterial pressures were still greatly elevated. Catheters passed high up the internal jugular vein showed that intrathoracic pressure changes were fully transmitted to the venous outflow of the brain. As can be seen in Fig. 1, the arterio-venous pressure difference across the brain may reach its lowest level during the paroxysm.

Intermittent Coughing

This was the "normal" form of coughing, inspiration following each cough in a fairly regular sequence. It caused large rapid fluctuations in intrathoracic pressure, which will be termed "cough transients." It was followed in all subjects by a fall of arterial pressure. An example of syncope from this form of coughing is shown in Fig. 2. Consciousness was lost at the lowest blood-pressure level.

Physiological Effects of Coughing

Fig. 3 summarizes the changes caused by the Valsalva manœuvre (or continuous coughing) and intermittent coughing. The data have been published elsewhere (Sharpey-Schafer, 1953). A to B is the period of raised intrathoracic pressure. This pressure is transmitted to the arteries, veins without valves, and the chambers of the heart. In the periphery, venous valves shut and blood accumulates distal to them at a rate which depends on previous vasomotor tone (Howard *et al.*, 1951) and the height to which the arterial supply pressure is raised.

The accumulation of blood in peripheral veins results in a decrease in the filling pressure of the heart and subsequent fall in stroke output. Pulse pressure is decreased in peripheral arteries during the Valsalva manœuvre or continuous coughing, but large cough transients are the conspicuous feature of arterial records in intermittent coughing. Baroceptors therefore are exposed to diminished pulsations during the Valsalva manœuvre and to large pressure transients during intermittent coughing. In the former case the response is peripheral constriction and in the latter dilatation, as shown in the forearm during the period B to D. These blood-flow changes do not occur in sympathectomized limbs, and are therefore mediated through the vasomotor nerves. The suggestion that these circulatory responses result from decreased or increased stimulation of baroceptors by rapid transients is supported by recent work on the isolated carotid sinus of the dog (Ead *et al.*, 1952).

There is a further important difference between the Valsalva manœuvre and intermittent coughing. In the former at point B the intrathoracic pressure falls immediately to the control level; in the latter it is elevated 10 or more mm. Hg (Fig. 3) and may remain so for many seconds during the period B to D. This raised pressure is transmitted to the chambers of the heart and great veins (Fig. 4) and may play a part in the production of post-tussive arterial hypotension by reducing cardiac output below control values, since it can be shown that positive-pressure breathing causes a fall in cardiac output (Werkö, 1947). There was no evidence on measurement of pulmonary arterial vasoconstriction as suggested by McCann *et al.* (1949).

Clinical Considerations

Some knowledge of the causes of this form of syncope has been helpful in the interpretation of symptoms. If normal physiological mechanisms only were involved it seemed unlikely that the condition was rare. The 27 cases investigated were found without difficulty over a period of two years and many others were seen clinically. Indeed, minor symptoms such as dizziness are almost universal among those who cough strongly. The curious rarity of the

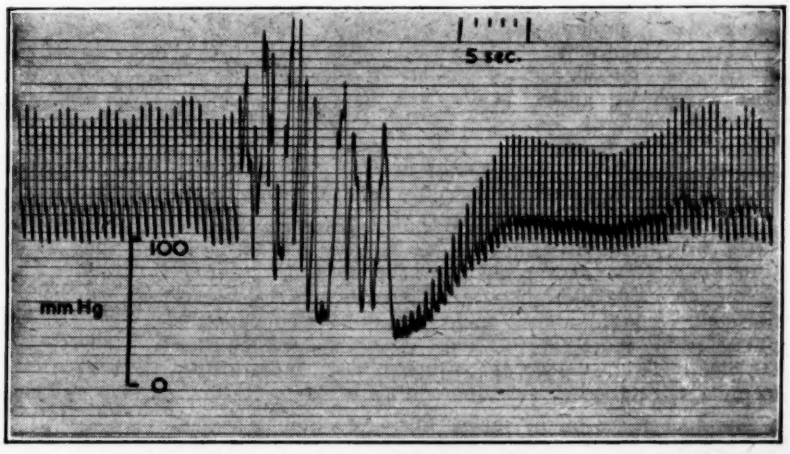

FIG. 2.—Arterial pressure record of intermittent coughing. Large arterial cough transients were followed by hypotension and syncope.

syndrome in females may be due, in part, to social taboos which impose the belief that such exhibitions of violence are "unladylike." The case of a Scots woman with typical attacks has, however, been reported to me. Continuous records are not yet available from children with whooping-cough and infants with breath-holding.

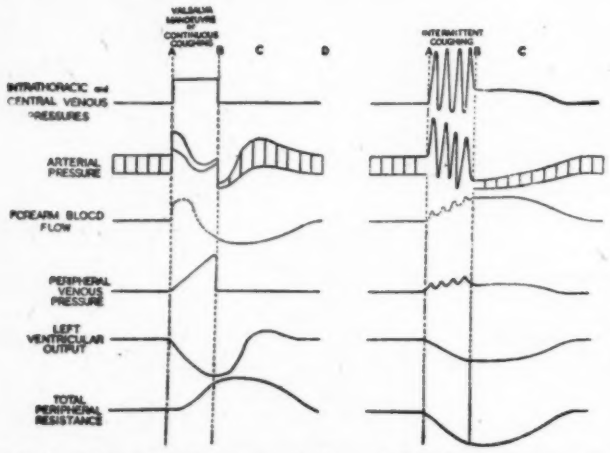

FIG. 3.—Diagrammatic representation of the circulatory events caused by the Valsalva manœuvre or continuous coughing, and intermittent coughing (see text). The changes in left ventricular output were not measured directly but were inferred from the arterial records.

Patients with large quantities of sputum from chronic pulmonary disease rarely show these symptoms. Continuous records during coughing in such patients showed that intrathoracic pressure rose no higher than about 100 mm. Hg, and although the usual circulatory changes were present they were not sufficiently pronounced to interfere with the blood supply to the brain.

The role of smoking, drinking, and bad teeth and other infections is to produce the upper respiratory tickle, which, like some forms of skin itching, needs the utmost violence to assuage. This form of coughing may become a habit. It is easy to see why middle-aged males are the chief victims.

A history of a single cough followed by immediate unconsciousness should be regarded with suspicion. Three of the subjects reported here gave such a history, but proved to have typical attacks of prolonged violent coughing followed by syncope. Questioned after a recorded attack, they still claimed immediate unconsciousness. Two may have had a short period of retrograde amnesia; the third interpreted 15 seconds as "immediate."

The occurrence of fits during syncope after coughing is likely to be more misleading to neurological than to circulatory workers, who are probably aware that a fit can occur in any normal subject if the blood flow to the brain is sufficiently reduced. If the subject is a known epileptic the full seizure is more easily induced by methods of lowering the blood pressure. Two of the present cases had a history of epilepsy and grand mal after violent coughing.

The appearance of the subject during continuous violent coughing is alarming, and may suggest to the lay and even the medical mind the likelihood of a burst blood vessel, while the subsequent syncope may indicate that this calamity has occurred. The vessels of internal organs such as the brain, however, are protected from high pressure differences across their walls, since surrounding structures are exposed to the same pressure, and, as explained above, the pressure across these vessels is less, not more, than normal.

It is only in places where veins are in near contact with the outside atmosphere that pressure differences of 300 mm. Hg may be built up. Since surface veins are protected by valves, high pressures are reached only after a period of some seconds. In prolonged paroxysms of coughing nose veins commonly burst, and occasionally veins in the conjunctiva and anus do likewise.

Treatment

Attempts to alter bad habits or causal irritant factors proved singularly unsuccessful in this group of middle-aged men. Explanation of the mechanism was tried with more intelligent subjects, but even these found it difficult to realize that extreme violence was unnecessary and clung to their way of coughing for various fanciful and erroneous reasons.

The investigation, however, showed a method which, while annoying to the investigator, was almost uniformly successful in alleviating the attacks. It was found on the first occasion when measurements were made that an attack could be induced with ease. Subsequently more and more encouragement became necessary to produce the required intrathoracic pressure changes. The subjects learnt by demonstration that it was violence which caused syncope. As one said on the fifth occasion, "I don't see why I should cough myself unconscious any more—to please you, Doctor."

Summary

Syncope from coughing was found to be common and to result from an acute diminution of supply pressure of blood to the brain. Continuous records in 27 cases indicated that normal physiological mechanisms only were involved. The violence of the coughing was the determining factor, intrathoracic pressures of 200 to 300 mm. Hg being reached.

Subjects who coughed continuously without drawing breath were shown to have circulatory changes which were exaggerated examples of the effects of the Valsalva manœuvre. Consciousness was usually lost during the period of acute hypotension following cessation of coughing. Occasionally syncope occurred while intrathoracic pressure was still high, but the arterio-venous pressure difference across the brain was greatly reduced.

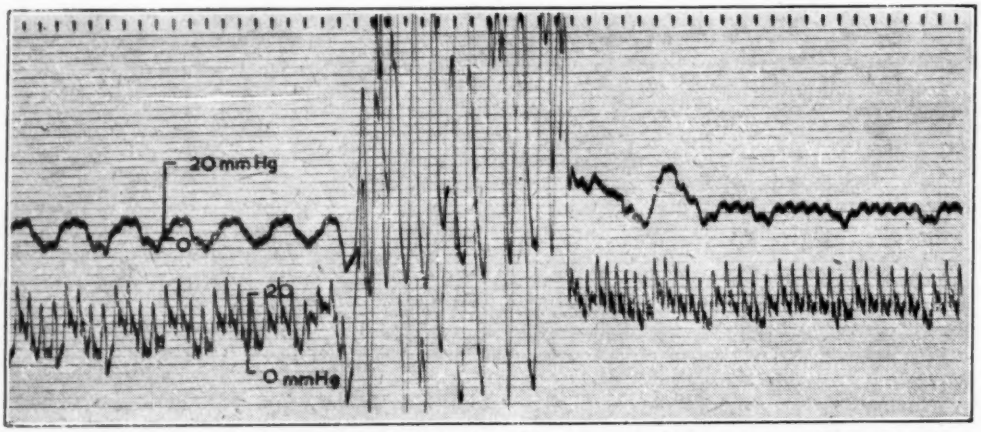

FIG. 4.—Upper curve, intra-oesophageal (intrathoracic) pressure; lower curve, pulmonary artery pressure. Both were elevated to the same extent after coughing.

Subjects who coughed intermittently also had syncope. Arterial pressure and peripheral blood flow records showed vasodilatation following this form of coughing in contrast to the vasoconstriction which resulted from the Valsalva manœuvre and continuous coughing.

It is suggested that vasodilatation following intermittent coughing is due to the stimulation of baroceptors by large arterial cough transients.

Intrathoracic pressure remained elevated about 10 mm. Hg for many seconds following intermittent coughing.

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EFFECTS ON BLOOD PRESSURE OF VENTRICULAR ASYSTOLE DURING STOKES–ADAMS ATTACKS AND ACETYLCHOLINE INJECTIONS

BY

E. D. BARLOW, M.B., B.Chir., D.P.M.

Research Fellow, St. Thomas's Hospital Medical School

AND

SHEILA HOWARTH, M.B., B.S.

Senior Research Fellow, Institute of Cardiology, National Heart Hospital

Ventricular standstill may occur spontaneously in cases of heart-block, and during Stokes–Adams attacks consciousness is lost if asystole is sufficiently prolonged. Cardiac arrest also follows the intravenous injection of acetylcholine, with resulting loss of consciousness (Phillips *et al.*, 1952). Observations have been made to determine the peripheral arterial pressure in these two types of case, and the results are reported in this paper.

Blood pressure was recorded continuously in the brachial artery, using a capacitance manometer (Hansen, 1949). The Stokes–Adams attacks occurred spontaneously in a patient with heart-block, who later showed no macroscopic heart lesion at necropsy. Rapid intravenous injections of 50 to 200 mg. of acetylcholine chloride were given as a part of routine therapy to psychoneurotic patients who had no organic circulatory abnormality on clinical examination.

Results

The blood-pressure records in both types of case show a striking similarity. Typical examples are shown in Figs. 1 and 2. Ventricular asystole was always followed by an acute fall in blood pressure. With prolonged standstill, blood pressure fell rapidly at first, then more slowly, and finally remained constant at about 20–25 mm. Hg. Loss of consciousness was often followed by convulsive movements.

When ventricular contractions were resumed an "overshoot" was seen on all blood-pressure records: systolic and diastolic pressures rose above control levels for a variable number of seconds before the blood pressure returned to normal. An increase in pulse pressure, more striking in the Stokes–Adams attacks, was recorded during the overshoot. The characteristic flush which accompanied restoration of the circulation following the Stokes–Adams attacks was also observed after cardiac arrest from acetylcholine injection.

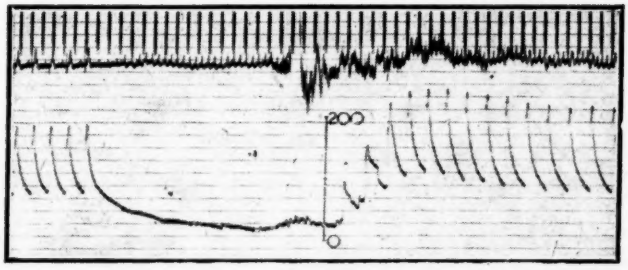

Fig. 1.—Effects on the blood pressure of a Stokes–Adams attack. Above: Lead II of the electrocardiogram. Convulsive movements produced interference with the record after 16 seconds asystole. Below: Continuous brachial arterial pressure record. Time (top) in seconds. Calibration in mm. Hg.

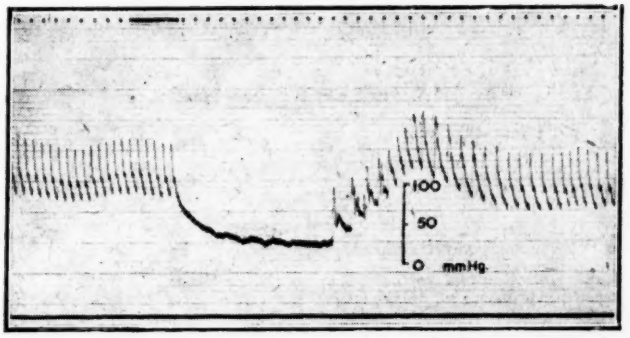

Fig. 2.—Effect on the blood pressure of cardiac arrest produced by 100 mg. of acetylcholine given intravenously. Period of injection is shown by signal on time marker. Time in seconds.

Discussion

In spontaneous and induced ventricular asystole, blood pressure falls rapidly to syncopal levels. Systemic arterial pressure tends to stabilize during cardiac standstill at 20–25 mm. Hg. This figure does not differ greatly from the standing pressure of 14 to 22 mm. Hg observed by Dowling *et al.* (1952) during asystole induced for 5 to 10 seconds by carotid sinus pressure. These authors, however, did not record a subsequent overshoot in the systemic pressure.

The overshoot may be regarded as a response to the lowered blood pressure. A similar overshoot follows the fall in effective systemic pressure during the Valsalva manœuvre (Hamilton *et al.*, 1936; Howard *et al.*, 1951), where it has been shown to be due to sympathetic vasoconstriction (Wilkins and Culbertson, 1947). Vasoconstriction has been demonstrated in muscle vessels during the overshoot of the Valsalva manœuvre (Howard *et al.*, personal communication); but vasodilatation may be present in the vessels of the skin during the overshoot after asystole, since vivid flushing of the face and neck occurs in both Stokes–Adams attacks and after intravenous acetylcholine injection.

The striking similarity between acetylcholine-induced asystole and spontaneously occurring Stokes–Adams attacks suggests that these effects of intravenous acetylcholine injections result from asystole alone.

Summary

During ventricular asystole in spontaneously occurring Stokes–Adams attacks and following rapid intravenous injections of acetylcholine systemic blood pressure fell to low levels.

During cardiac standstill of up to 21 seconds arterial pressure remained constant at 20 to 25 mm. Hg.

An "overshoot" occurred in the blood pressure following the resumption of ventricular contractions in both forms of asystole : systolic and diastolic pressures increased above control levels before arterial pressure returned to normal.

It is suggested that this action of intravenous acetylcholine results from asystole only.

The cases investigated were under the care of the physicians to St. Thomas's Hospital and the National Heart Hospital.

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HIATUS HERNIA AND PREGNANCY

A REVIEW OF NINE CASES AND THE LITERATURE

BY

W. A. W. DUTTON, M.B., M.R.C.S.
D.Obst.R.C.O.G.

AND

H. J. BLAND, M.B., Ch.B., D.Obst.R.C.O.G.
Formerly Resident Obstetric Officers, Southmead General Hospital, Bristol

Herniation of part of the stomach through the oesophageal hiatus of the diaphragm is a fruitful source of errors in diagnosis. Although this disorder is being detected more often (Avery Jones, 1952), it is still commonly overlooked.

Hiatus hernia is probably due in most cases to factors operating in adult life. Laxity of the tissues, with deficiency at the oesophageal ring, is common (Harrington, 1945). Hernia at this site is favoured by conditions raising intra-abdominal pressure and by recumbency. Such herniae may be oesophago-gastric or para-oesophageal, termed by Allison (1951) the sliding and rolling types, which may be present together.

The symptoms are protean, and have been well described by Nicholson (1952). Epigastric discomfort and vomiting are common. If the disorder is complicated by reflux oesophagitis there may be intense "burning pain" behind the lower end of the sternum, which may radiate up to the neck. There may be slight dysphagia, and a gastric ulcer may develop near the cardia. Retching of blood-stained fluid may proceed to frank haematemesis and melaena. Stricture may develop at the lower end of the oesophagus.

Review of Literature

Reports of hiatus hernia complicating pregnancy are infrequent. All authors emphasize that symptoms are aggravated by recumbency, worsen in the last trimester, and tend to clear up after delivery. Heartburn is particularly constant. Evans and Bouslog (1940) reported four cases in which this symptom was almost intractable, and Bush (1950) reported a similar case in which radiation of the pain to the ears was a bizarre addition. Vomiting may be severe and often culminates in haematemesis, which has been described by Schnepp (1943), Queries and Minor Notes (1937), Bourgeois and Hood (1949), Simmons *et al.* (1950), and Hahn (1952). Murless (1947) reported two cases of large para-oesophageal herniae causing vomiting and dyspnoea in pregnancy. Penman (1951) added to the literature four cases in which hiatus hernia produced persistent gastro-intestinal disturbances during pregnancy.

Vinson (1921, 1923) described nine cases of oesophageal stricture following severe vomiting in pregnancy. Some of the records of these cases suggest that they were due to hiatus hernia. Rennie, Land, and Park (1949) described five cases of pregnant women who developed dysphagia and retrosternal pain in the latter months of pregnancy. Hiatus hernia associated with a short oesophagus and stricture was present in all five cases. In three, active oesophageal ulceration was demonstrated. Allison, Johnstone, and Royce (1943) described a similar case.

Case Reports

The following cases came to our notice during routine duties in 1950–2.

Case 1.—The patient, a 2-gravida, 1-para aged 31, was a thin woman whose previous health and first pregnancy were entirely normal. Her second pregnancy (estimated date of delivery, October 2, 1951) was complicated. Morning nausea and retching began at the sixth week and persisted throughout pregnancy. During the second trimester there were repeated attacks of "burning" pain behind the xiphoid, exacerbated by meals, recumbency, and stooping forward. Symptoms were worse at night and during the third trimester. Vomiting became so frequent that she was admitted to hospital at the thirty-eighth week and treated symptomatically. Her condition fluctuated until September 24, when, with the spontaneous onset of labour, vomiting culminated in a haematemesis. Labour was inert, and copious vomiting resulted in dehydration and ketosis. Intravenous fluids and gastric aspiration were begun, but a second haematemesis occurred which required blood transfusion. After spontaneous rupture of the membranes her condition improved and normal delivery followed. During the puerperium there were no untoward symptoms. On the ninth day a barium-meal examination showed a small para-oesophageal hernia without oesophageal ulceration or stricture. The stomach and duodenum were normal. We have been unable to trace the patient for a follow-up.

Case 2.—A 4-gravida, 3-para aged 32 attended the antenatal clinic at 20 weeks ; the estimated date of delivery was January 8, 1952. She was a thin woman and complained of nausea, heartburn, and retching dating from the first month. No abnormality was found on clinical examination. At subsequent visits she reiterated these symptoms, for which a variety of alkaline medicines gave little relief. During the last trimester she emphasized that the heartburn was much worse at night, was better when she got up to vomit, only to recur when she lay down. From the thirty-eighth week the pain was noted to radiate up to the neck, and there was occasional dysphagia. On January 4 she vomited a few ounces of bright-red blood and was admitted to hospital. She responded fairly well, being nursed propped

up and given a light diet. Vomiting was only occasional, and labour on January 8 was uncomplicated, as was the puerperium. On the seventh day a barium-meal examination revealed a simple sliding hiatus hernia. The stomach and duodenum were normal. When seen in September, 1952, she had very occasional heartburn. A barium-meal examination was entirely normal.

Case 3.—A 2-gravida aged 40 was admitted to hospital on April 2, 1951, for a gynaecological operation. On routine questioning about her pregnancies, she stated that during the first pregnancy, 13 years previously, she had suffered from severe heartburn and vomiting from the sixth week. From the seventh month she had had to sleep propped up, so severe was the pain when she lay down. Symptoms cleared up with the onset of labour ("I never had a sick labour"). The second pregnancy, five years later, was "like a repetition of the first." After her pregnancies she kept fairly well until she became obese. In recent months she had experienced several attacks of heartburn, "indigestion," and vomiting relating to posture, being worse when lying down or stooping. She sometimes vomited "lumps like liver." A barium-meal examination revealed a large Type I hiatus hernia with marked regurgitation of stomach contents when in the prone position. Reduction and repair of the hernia was undertaken in the thoracic unit of another hospital. She has since been symptom-free, and was last seen in September, 1952.

Case 4.—A primipara aged 25 was admitted to hospital as an emergency case on February 5, 1952. She was 36 weeks pregnant. Her previous health had been normal. She had had slight morning sickness from the first month of pregnancy. From the fifth month she suffered from recurrent "burning" substernal pains, especially when prone at night, with occasional small vomits. During the month before admission the vomiting, though more frequent (sometimes ten times a day), was not copious. The patient was nursed propped up in bed. A normal diet was fairly well tolerated, but heartburn and vomiting recurred at intervals. A barium-meal examination on February 13 showed a Type I hiatus hernia, without complications. By February 20 the vomiting had become more persistent, and, after failure of medical induction, surgical induction of labour was undertaken. The progress of labour was unsatisfactory; vomiting became very severe, but there was no haematemesis. Dehydration and ketosis necessitated intravenous fluids, and labour was terminated after two hours in the second stage, by episiotomy and low forceps extraction under local analgesia. The puerperium was uncomplicated. She was well and symptom-free in May, 1952.

Case 5.—A primipara aged 25, 36 weeks pregnant, was admitted to hospital on February 15, 1952, as a case of rheumatic heart disease. She had been well until about 20 weeks, when she began to suffer from heartburn, retching, and occasional vomiting, especially at night, relieved by sitting or standing. Between the twenty-eighth and thirty-sixth weeks her exercise tolerance deteriorated, she became orthopnoeic, and she slept propped up at night. Henceforth, the heartburn, retching, and vomiting concerned her much less, although still present. On February 2 chest screening was undertaken, and a barium swallow confirmed enlargement of the left antrum and, concurrently, a small Type I hiatus hernia. Caesarean section under epidural anaesthesia was performed because of severe pre-eclampsia. There were no puerperal complications. A barium meal in September, 1952, failed to demonstrate a hiatus hernia.

Case 6.—A 2-gravida, 1-para aged 34, whose previous health had been good, was admitted as an emergency case on February 2, 1951, being 28 weeks pregnant. There had been severe vomiting during the first trimester in both pregnancies. The second pregnancy was further complicated by epigastric pains, which were particularly severe as pregnancy advanced. Vomiting was almost continuous for four days before admission, when she was noted to be thin and dehydrated, with ketonuria. The vomit was blood-stained. She was nursed sitting up in bed, and given parenteral fluids and a continuous intragastric milk drip. A barium meal on February 5 showed a large Type I hiatus hernia without complications. She was discharged home symptom-free, and was subsequently delivered at term at another hospital. In July there was still slight indigestion, and a barium meal showed that the hernia was still present but was much smaller.

Case 7.—A primigravida aged 23 was admitted as an emergency case on December 7, 1950, when 35 weeks pregnant. There had been slight nausea and vomiting during the first trimester, and then no complaint until heartburn and vomiting began at 20 weeks. Symptoms were worse when lying down at night, and were relieved by sitting up. After a severe bout of vomiting, she was admitted to hospital. Dehydration required drip therapy, and she was discharged symptom-free on the seventh day. Symptoms recurred, but were mild until January 11, 1951, when she was admitted in labour. Labour was inert, there was continuous post-xiphoid pain, and frequent vomiting of small amounts of stale blood occurred. Intravenous fluids were given, and normal delivery followed. The puerperium was uncomplicated. In May she complained of dysphagia and had lost 28 lb. (12.7 kg.) in weight. A barium meal showed a hiatus hernia with an oesophageal stenosis immediately above it, about 2 cm. in length. This was later confirmed by oesophagoscopy, during which the oesophagus bled easily. The stricture was subsequently successfully resected.

Case 8.—A 2-para aged 40 had her first pregnancy in 1931. It was characterized by severe vomiting throughout and severe nocturnal post-xiphoid pains related to posture. Vomiting during labour had been severe, but was absent in the puerperium. The second pregnancy, in 1943, was like a repetition of the first. In recent years she had become plump, and the post-xiphoid pains had returned, being worse at night and on bending forwards, but were relieved by alkalis, vomiting, or sitting up. A barium meal in July, 1951, revealed a fairly large hiatus hernia.

Case 9.—A primigravida aged 24 was admitted on September 25, 1950, complaining of pains in the right iliac fossa, and persistent nausea and vomiting. Laparotomy revealed a 16 weeks pregnancy; a simple cyst of the right ovary was removed, together with a normal appendix. She remained well for six weeks, then began to suffer from severe nausea and vomiting, especially when lying down and after meals. On readmission on March 3, 1951, she was dehydrated, and 24 hours later she had a small haematemesis. Persistent vomiting necessitated almost continuous intravenous fluids. A barium-meal examination on March 8 revealed a Type I hiatus hernia with probable oesophageal ulceration. Surgical induction of labour was undertaken, but the first stage was inert, and she continued to vomit blood-stained fluid. As the maternal condition was deteriorating labour was terminated by caesarean section after 48 hours. A barium swallow on the twelfth post-operative day revealed marked functional improvement of the cardia, with little tendency to regurgitation of barium. A small tubular hiatal protrusion was still present, but only in the optimum position. There was no evidence of ulceration. In May, 1952, the patient was still complaining of nausea, heartburn, and occasional vomiting.

Discussion

Rigler and Eneboe (1935) have shown that the association of hiatus hernia with pregnancy is common, but they were unable to demonstrate any correlation between gastric symptoms and the presence or absence of hernia.

On consideration of the manner in which hiatus hernia can produce symptoms these findings are unexpected. It is possible that hiatus hernia in pregnancy is often overlooked for a variety of reasons. In current teaching concerning vomiting in pregnancy, neurosis is often cited, and some of the symptoms of hiatus hernia do suggest a functional illness. Further, the vomiting caused by hiatus hernia frequently ceases when the patient rests sitting up in bed, and

may cease to vomit throughout her stay in hospital, thus testing the credulity of her medical attendant.

A radiologist needs skill and experience to demonstrate a hiatus hernia. Resolution is rapid in the puerperium, and attempts to make a post-partum diagnosis may be unsuccessful because of the diminution in size of the hernia, or even its disappearance. Rigler and Eneboe (1935), Bourgeois and Hood (1949), and Schnepp (1943) all commented on this feature, our experience of which has been similar.

It is not intended to claim that hiatus hernia always causes trouble in pregnancy, or that such a hernia is always responsible for heartburn, vomiting, or haematemesis. We have seen cases illustrating the contrary.

It would seem that there is a need for a large-scale investigation to ascertain the role of hiatus hernia in heartburn and vomiting of pregnancy.

Summary

The association of hiatus hernia with pregnancy is described.

Nine cases with symptoms attributed to a hiatus hernia in pregnancy are reported.

The literature is briefly reviewed.

The need for further investigation is recognized.

We wish to thank Dr. J. M. Naish, Mr. W. M. Capper, Mr. P. Phillips, and Professor H. J. Drew Smythe for permission to publish these cases, Professor G. Gordon Lennon for helpful advice and criticism, and Dr. G. R. Airth, who performed most of the *x*-ray examinations.

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In his annual report the Medical Director of the Central Middlesex Hospital, Dr. Horace Joules, states that despite efforts to reduce the surgical waiting-list it still stands at well over 1,000. This number had been somewhat lessened by the co-operation of several other hospitals which had taken over large numbers, but last winter's fog and the subsequent high incidence of influenza had caused such a demand for acute admissions that the number waiting for surgery had risen to its previous height. Dr. Joules said that it was known that several London hospitals had practically no waiting-list, and he suggested that the time had come to evolve some system of transfer of cases for elective surgery between one hospital and another. A committee had been set up to inquire into the out-patient position, and had agreed that any further increase in numbers must be avoided. Closer association between practitioners and the hospital had been maintained by meetings of local health liaison committees and clinical meetings and courses. The opening of a new mass *x*-ray unit, domiciliary visiting, and the provision of additional beds had ensured early treatment of new cases of pulmonary tuberculosis. The association with the Middlesex Hospital had been strengthened by an agreement to continue undergraduate teaching at the Central Middlesex Hospital for a further 10 years, and by a scheme to exchange senior registrars after they had two years' experience in their own hospital.

TREATMENT OF TETANUS

BY

J. H. McINTYRE, M.D.

Grey Hospital, Greymouth, New Zealand

In an article on tetanus successfully treated with gallamine triethiodide ("flaxedil") Smith and Thorne (1952) advocate the use of this drug. They state that relaxation is secured on a steady level and that respiratory depression is less marked with gallamine triethiodide than with D-tubocurarine, especially in the conscious subject. It is to be noted, however, that the period elapsing between the infecting injury and the onset of symptoms in their case was at least ten days, so that the prognosis would be relatively good.

Recently we have had in this hospital two cases of severe tetanus in children in which the periods elapsing between injury and the onset of symptoms were under a week, being in one case four days and in the other six days. Both of these children died, one in 24 hours and the other in three days, despite treatment with gallamine triethiodide and the usual treatment with sedation, antitoxin, and antibiotics. Gallamine was not conspicuously successful in either of our cases in relieving spasm, although used in much the same doses as in the case of Smith and Thorne, and, in addition, we found it apt to cause respiratory embarrassment.

Case 1

A girl aged 4 was admitted to Grey Hospital on December 12, 1952. The mother stated that the child had cut her left thumb in the garden on some glass six days previously. She now complained of neck stiffness and was unable to open her mouth. On the morning of her admission facial spasm became apparent. On examination the pulse was 120 and the temperature 98.4° F. (36.9° C.), although this rose to 102° F. (38.9° C.) within a few hours. The mouth could not be opened more than about 0.5 cm. Risus sardonicus was present. Some stiffness of the muscles of the back was apparent, and within an hour of admission two spasms occurred. On the child's left thumb a deep infected laceration was seen.

A diagnosis of tetanus was made and 20,000 units of antitoxin was given, followed by a further 60,000 within six hours. Crystalline penicillin in doses of 500,000 units, later increased to 1,000,000, was given six-hourly. As further spasms were occurring an intravenous saline drip was set up and 5 mg. of gallamine was given intravenously and 20 mg. intramuscularly. This caused a temporary cessation of respiration, and mucus had to be removed with a suction apparatus. The pulse remained steady throughout this episode. Oxygen was given by nasal catheter, and the child recovered sufficiently to talk. About one hour later two further tetanic spasms occurred, which necessitated the addition of 1 ml. of thiopentone to the drip, and gallamine was required. Despite this, several convulsions occurred. As additional sedation 30 ml. of alcohol was introduced into each vacoliter, limited to two vacoliters daily. On the third day (December 15) the child had an exceptionally severe convulsion and died. Immediate restorative measures failed, as also did cardiac massage for one hour. Throughout, the child was "specialled" by the medical staff.

A total of 27 ml. of thiopentone and 350 mg. of gallamine was given in the three days that the child lived. In addition 4.5 ml. of paraldehyde was also given, and intravenous alcohol. Despite this treatment she had convulsions of more or less severity, on an average twice or three times an hour. Respiratory embarrassment occurred seven times, and on

two occasions artificial respiration was necessary. Oxygen was often required. Mucus in the air passages was very troublesome and required frequent suction.

Some 180 mg. of gallamine was given intramuscularly and 170 mg. intravenously; the doses varied from 5 mg. intravenously to 20 mg. intramuscularly. *Clostridium tetani* was grown from swabs taken from the wound when it was cleaned.

Case 2

On January 22, 1953, a boy aged 7 was admitted to Grey Hospital. He was quite rigid, was unable to open his mouth, and was perspiring freely. He complained of abdominal pain, and the muscles of the back were very rigid and in spasm. His parents gave a history of his having jumped on to a spike of wood in the grass four days previously. The wound had healed. The boy was quite conscious; his temperature was 99.2° F. (37.3° C.), and pulse 140. He was given 40,000 units of antitoxin, and as clonic spasms were almost continuous a slow intravenous saline drip was set up and 40,000 units of antitoxin and 80 mg. of gallamine were introduced into each vacoliter. In addition 1,000,000 units of penicillin was given four-hourly. Thiopentone, 7 ml., was given as an initial sedative into the tubing of the drip. A dose of 80 mg. of gallamine in the first vacoliter and 40 mg. in subsequent vacoliters was decided upon. Later it was found necessary to give additional gallamine intravenously, the dose being relatively small. Later it was found necessary, however, to increase the dose of gallamine to 80 mg. and finally to 120 mg. in each vacoliter.

Atropine sulphate, 1/200 gr. (0.32 mg.) intravenously, had previously been administered, but even so his breathing became very bubbly. A further 1/200 gr. (0.32 mg.) of atropine was given. For the first few hours the convulsions were relatively few, but as time went on they increased in number and severity, so that 10 hours after admission he was having almost continuous clonic convulsions, and anaesthetic doses of thiopentone were required. Although he was in hospital for only some 23 hours before he died, he had numerous mild and many severe spasms. A total of 29.5 ml. of thiopentone and 404 mg. of gallamine was given intravenously in an effort to suppress his convulsions. This in addition to antitoxin, 80,000 units, and penicillin, 1,000,000 units four-hourly. Although his breathing was very bubbly, respiration was not arrested and no artificial respiration was required. The boy died the morning after admission, death being due to sudden heart failure.

Discussion

These two cases do not make impressive reading. They tend to bear out the axiom that tetanus occurring within a week of injury is invariably fatal. In the first instance we had the impression that gallamine caused some respiratory embarrassment, but this was not confirmed in the second patient, who was, however, desperately ill on admission.

Abel (1934), in a study of the path of conduction of the toxin to the central nervous system, indicates that this occurs by way of the blood stream. He also suggests that the tetanus toxin is altered into or liberates in the cord a substance which has a lethal effect on the vital centres.

The dose of sedatives could not be reduced in either of these two cases without causing severe suffering. Penicillin, even in doses of 1,000,000 units four-hourly, did not seem to affect the course of the disease. Antitoxic serum in doses of 80,000 units in the first twelve hours likewise seemed to have little effect.

It can only be concluded that the present methods of treatment of tetanus in cases occurring within a week of injury are ineffective, and that, in view of the fact that 35 cases of tetanus occurred in New Zealand last year, recourse should be had to the routine active immunization of all children against tetanus.

I bring these cases to the notice of the public health authorities, both in the United Kingdom and in New Zealand, as so often in minor injuries one is in doubt whether the injury requires a prophylactic injection of antitoxin or not. In view of these two fatal cases it would seem to be worth while to include antitetanic inoculation at the same time as routine immunization against diphtheria is being carried out.

My thanks are due to Dr. B. M. Dallas, Dr. E. E. Tilson, and Mr. S. Barclay, the surgeon superintendent—the last-named for his efforts at resuscitation of Case 1. I also thank Mr. B. Heine and Mr. R. D. Lockhart for their constant attention to these cases.

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Medical Memoranda

Stasis of the Large Bowel Secondary to Acute Cholecystitis

It is generally recognized that acute inflammation of the gall-bladder may be accompanied by upsets of large-bowel function leading to distension and constipation; the extreme degree to which distension may proceed, simulating large-bowel obstruction, is worthy of greater emphasis than it usually receives, and is well illustrated by the following two cases.

Case 1

A married woman aged 72 had been operated on four months previously for a strangulated right inguinal hernia, and had made a smooth recovery. Two days before her present admission she had had an attack of vomiting, during which she had been examined at a London teaching hospital but had not been detained. She complained of abdominal pain of two days' duration which was situated mainly in the right side of the abdomen. There was anorexia and she had passed neither faeces nor flatus for three days.

On examination her temperature was 101° F. (38.3° C.), pulse 110, and blood pressure 130/80. The tongue was furred. The abdomen was distended, with maximal and marked distension in the right flank; the caecum was palpable, and above this was a tender mass. Rectal examination was negative. A plain *x*-ray film of the abdomen showed much distension of the caecum and ascending colon. The diagnosis was thought to lie between an attack of acute cholecystitis and a perforated carcinoma of the ascending colon; in view of the attack of vomiting two days earlier the former was considered more likely.

Operation.—A right paramedian incision was made. The caecum and the ascending colon were distended to at least twice their normal size, but there was no organic obstruction. The distension extended to that portion of the ascending colon which lay in contact with an acutely inflamed distended gall-bladder; gall-stones were present. Cholecystectomy was performed and the abdomen closed with drainage. The patient made an uneventful recovery.

Case 2

A married woman aged 80 had had attacks of abdominal discomfort associated with distension and relief by the passage of flatus during the past year. She had had abdominal pain for three days; the pain was mainly in the lower abdomen and was described as being "windy" in character. She was constipated and had not passed flatus for three days. Her abdomen felt as if it had become "blown-up." There had been some retching but no vomiting.

On examination her temperature was 98° F. (36.7° C.), pulse 112, and respirations 24. The tongue was moist.

The abdomen was tensely distended, maximally in the subumbilical zone, which was very prominent, tight, and tympanitic. Some degree of tenderness was present in the midline below the umbilicus. Intestinal sounds were heard. The release phenomenon was absent and Murphy's sign was negative. There was no jaundice. Some constipated faeces were found in the rectum; there was no pelvic tenderness. A plain *x*-ray film of the abdomen showed distension of the large bowel round to the sigmoid colon distally. The suspicion of an inflammatory lesion was aroused, but in view of the negative Murphy sign it was thought that the appendix would be the seat of the inflammation if the diagnosis of large-bowel obstruction was proved incorrect and an inflammatory lesion was present.

Operation.—A right paramedian incision was made. The large bowel was distended as far as the sigmoid colon, but there was no organic lesion in it. The gall-bladder was acutely inflamed; an area close to its fundus was near to gangrene. Cholecystectomy was performed. The gall-bladder contained numerous small pigment calculi. The abdomen was closed with drainage.

Comment

The extreme degree of abdominal distension made this the most prominent physical sign in each of these cases of acute cholecystitis. The presence of marked distension together with constipation in patients aged 72 and 80 years makes one incline towards a diagnosis of large-bowel obstruction. In Case 1 the correct diagnosis was considered, but in Case 2 it was excluded as Murphy's sign, which was specifically tested, was negative. In this case the degree of distension was quite remarkable and was not regarded as likely to be produced by anything save mechanical obstruction, especially as intestinal sounds were heard on auscultation.

Most of the standard textbooks of surgery make passing mention of bowel distension and constipation when discussing acute cholecystitis, but the extreme degree, with its misleading implications, to which this may proceed is not emphasized enough. Kiely (1949), however, includes intestinal obstruction in his differential diagnosis of acute cholecystitis. The larger books naturally make more mention of the condition. Aird (1949) writes: "There is a flatulent distension of the right side of the abdomen after 36 to 48 hours (Wilkie), and sometimes the whole right side is tense." Maingot (1948) also mentions distension of the caecum and ascending colon; his further comment, "While in others the ballooned caecum may suggest a diagnosis of acute retro-caecal appendicitis or obstruction of the large intestine," is particularly apposite in regard to Case 2, in which these diagnoses were considered to the exclusion of that of acute cholecystitis. Finally, Morley (1947) writes: "Owing to the reflex inhibition of intestinal movements there is constipation and considerable distension, most marked in the colon."

Gordon Ferguson, F.R.C.S.,
Senior Surgical Registrar, Hastings Hospital Group.

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Comparison of the Male Toad and Hogben Pregnancy Tests

Numerous papers have been published showing the reliability of pregnancy tests on toads, but none which compared the Hogben test with the male toad test. Although male toad tests are in common use in other countries there seems to be an impression in this country that it is less accurate than the Hogben test. The figures given below suggest that it is equally reliable and has several advantages.

Methods.—Early morning urine is concentrated by Scott's method (1940). Then 2.5 ml. of the concentrate is injected subcutaneously into a female *Xenopus laevis* toad and 2 ml. into a common British male toad (*Bufo bufo*). Three hours after injection urine is obtained from the cloaca of the male toad with a blunt Pasteur pipette and examined microscopically for spermatozoa. One male toad is tested. The jar containing the *Xenopus* toad is inspected the following morning for eggs. If the result is negative a second toad is injected and the test read on the following day. If this result is positive the negative result on the first toad is discounted (Landgrebe and Hobson, 1949).

Results

Both Hogben and male toad tests positive ..	181
„ „ „ „ „ .. negative ..	200
Negative Hogben, positive male toad test ..	19
Positive Hogben, negative male toad test	0

In over 95% of cases both tests gave the same result, and in no instance was the Hogben test positive and the male toad test negative. Of the 19 cases in which the male toad test was positive and the Hogben test was negative, 18 proved to be pregnant. Seven were early pregnancies, and the Hogben test later became positive. Two were late pregnancies, in the fifth month. One case had a history of several miscarriages, but did not abort in the present instance. Eight patients aborted a few days after the test. The nineteenth patient was not pregnant, but had a fibroid; the Friedman test was negative; after removal of the fibroid the male toad test was negative. A Friedman test was done on 6 of these 18 cases, and was positive in all.

The results strongly suggest that the male toad test can detect a lower level of gonadotrophin in the urine than the Hogben test, as shown in very early pregnancy, late pregnancy, and impending abortion, in all of which conditions the urinary gonadotrophin is probably low. The number of impending abortions detected by a comparison of the tests is striking.

Advantages of the Male Toad Test

1. Comparison with the Hogben test shows it to be equally accurate and, in fact, more sensitive.

2. The test can be read in three hours. The Hogben test takes 24 hours, and if the first toad is negative a result cannot be given for 48 hours.

3. Only one toad is required. In a random series of 200 Hogben tests performed in three months in this laboratory, 16 gave a negative result on the first toad and a positive result on the second. That is, an error of 8% would have occurred by using one toad only.

4. Male toads are useful for dilution tests for a suspected hydatidiform mole. Half a dozen toads can be injected at the same time with different dilutions, read after three hours, and the appropriate dilution injected into a rabbit on the same day. The unreliability of the reaction of the *Xenopus* toad and the time it takes to react compare unfavourably with the male toad for this purpose.

5. The cost and trouble of maintaining male British toads are much less than for *Xenopus* toads. Their initial cost is much lower and the necessary equipment much cheaper. The care of the male toad is simple (Law, 1949). As only one toad is used for a test, and it can be used again within a week if necessary, a much smaller stock of male toads than of *Xenopus* toads is required for the same annual number of tests. The male toad pregnancy test is within the scope of the smallest laboratory.

I wish to acknowledge my gratitude to Dr. M. Haines and Dr. H. P. Ferreira for demonstrating the technique of the male toad test.

G. M. G. Barton, M.B., B.S.,
Pathologist, Salisbury Infirmary.

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Reviews

SURGICAL TREATMENT OF CHILDREN

The Surgery of Infancy and Childhood: Its Principles and Techniques. By Professor R. E. Gross, M.D., D.Sc. Drawings by Etta Piotti. (Pp. 1,000; 1,488 illustrations and 567 figures. 80s.) Philadelphia and London: W. B. Saunders Company. 1953.

This is a most important and useful book—and incidentally, as prices go nowadays, remarkably good value for money with its great size and lavish illustrations. No surgeon who treats children should be without it. In it are recorded the methods and results of one who has unrivalled experience in certain directions, and who in addition is one of the greatest of surgical technicians. It is an extremely personal work, the author giving his own opinions and experiences, seldom wasting time in quoting those of other people. The writing is simple and clear, without that polysyllabic pomposity that is the besetting fault of so much American medical writing. There is a notable absence of vague and oracular statements and of unsubstantiated opinions: in fact, the only ones I have noted are in the very small part of the book not written by Dr. Gross himself. The book is inevitably incomplete, since no man can cover the whole of surgery, even if limited to one age group. There is nothing about orthopaedics, about the nervous system, about hare-lip and cleft palate, tonsils and adenoids, or lymphatic glands. This must have caused the author some difficulty in deciding on a title; a difficulty which another great individualist, H. O. Thomas, solved by putting out much of his work as "Contributions to Medicine and Surgery."

On the surgery of the digestive and circulatory systems of the young this book is at present supreme. It is so because of the combination of Dr. Gross's qualities and the American habit of hustling special cases to special centres, in contrast to the British one of treating them locally. The really remarkable results set out could be obtained in no other way than by this concentration of cases under the care of a highly specialized and skilled team. The opinions of Dr. Gross on his own situation are not very easy to make out. He is against adding paediatric surgery to the number of officially recognized specialties; and when we consider the closed-shop mentality and the rather bogus mystique that characterize some of them we see his point. On the other hand, he considers that "our larger communities and many of our academic centres can support a group who are particularly interested in these problems." If this means that there should be paediatric surgeons, but not many of them, and that they should try to set a standard rather than establish a monopoly, the reviewer cordially agrees.

There is a short but sound section on anaesthetics, in which it is interesting to note that the fashionable use of muscle relaxants is strongly condemned and the opinion given that they have no place in the surgery of the young.

Excellent as this book is, there is no doubt that it would be improved by taking notice of work done outside the United States on the rapidly developing section of surgery of which it treats. Those who work elsewhere on similar lines must feel a longing to raise many questions and discuss many points. What, for instance, is the explanation of 20% of failures in that simple ligation of the ductus arteriosus which is found so satisfactory in Britain? Is the reason for not using the "intussusception-prolapse" technique, which we find cuts an hour off the time of recto-sigmoidectomy for Hirschsprung's disease, ignorance of the technique published in England long before the much clumsier device of Hiatt to which the author gives a reference?

If they are not unaware of what is happening in England and Sweden, how is it that in Boston they are satisfied to give a sufferer from hypospadias the power of urinating normally only at the age of 15 years or more? Is there any objection to the conception of the "covered anus" and the "ectopic anus," which workers here have found so helpful in classifying and treating deformities in this region? Dr. Gross, as a result of much investigation, holds that hormone therapy is useless in treating undescended testicles, but would not this opinion be strengthened by reference to the anatomical work on which it can be securely based? And on the subject of anatomy, why not set out exact indications for the extent of Rammstedt's operation, which may not be needed by anyone of the experience of Dr. Gross, but which can be very helpful to the less expert?

I have read this book with great admiration and benefit—but, it must be admitted, with occasional irritation at its insularity.

DENIS BROWNE.

SPLENOMEGALY

Hypersplenism and Surgery of the Spleen. By William Dameshek, M.D., and C. Stuart Welch, M.D. (Pp. 84; illustrated. $10.) New York: Grune and Stratton, Inc. 1953.

This book is highly original in its presentation, content, type, illustrations, and binding, but it is a convenient book to handle and a most valuable one to browse through. Its pages are of card and each page gives a diagram or table with a few explanatory phrases. It is really a bound pictorial demonstration of the American Medical Association exhibit on the same subject which was awarded the Billings Silver Medal in 1950. The authors clearly and succinctly demonstrate the anatomy, histology, and physiology of the spleen, and diagrammatically present the evidence for splenic hormones. They list in a table of names and dates the historic development of our understanding, such as it is, of splenic anaemia and other forms of splenomegaly, and they detail and criticize in the clearest possible fashion the nomenclature of those splenomegalies which are accompanied by cytopenia. The symptoms of hypersplenism, as the authors interpret the term, and the results of treatment, with diagrams and coloured photographs of the operation, are presented in the same tabular way, and the authors also consider haemolytic anaemia and thrombocytopenic purpura. There is a fully classified list of the causes of splenomegaly and the method of investigation of splenomegaly is covered too.

Having read the book, one feels that one has attended a lecture, beautifully illustrated by lantern slides, but given by an inaudible lecturer. I have the impression that it would be a difficult book for a student or a general practitioner to study with the purpose of learning about the diseases of the spleen that it covers, but it is an admirable synopsis of facts about the spleen and would provide valuable lecture illustrations for a teacher of undergraduates.

The chief interest of the book is in the technique of its presentation. Anyone interested in medical books or medical education should try to find access to it (its cost is 10 dollars). Among other attributes it possesses that of showing how a complicated case history can be reduced to 100 words and a diagram without loss of accuracy or detail.

IAN AIRD.

CHEMOTHERAPY

The Sulphonamides and Antibiotics in Man and Animals. By J. S. Lawrence, M.D., M.R.C.P., and J. Francis, M.Sc., M.R.C.V.S., with the assistance of A. Sorsby, M.D., F.R.C.S., and P. G. Scott, F.R.C.S. Second edition. (Pp. 482; 38 figures. £2 2s.) London: H. K. Lewis. 1953.

This book is described as a second edition, but since it was previously Lawrence's *The Sulphonamides in Theory and Practice* it is better regarded as a new book. It follows the usual plan of describing first the properties of individual drugs and then discussing their use in the treatment of different kinds of disease. The earlier chapters on the sulphonamides are excellent, and together with the glossary of trade names in the appendix will enable the reader to sift the truth from the present-day flood of advertising matter in this connexion. On the other hand, these drugs are overdone in some parts of the text. It is surely

not worth while nowadays to devote nearly five pages to the sulphonamide treatment of otitis media. They also figure largely in the section on peritonitis, which cites no authority later than 1947.

It must be difficult, particularly for four authors in Manchester, Brisbane, London, and Exeter respectively, to produce an up-to-date book on so rapidly developing a subject, and, although isoniazid has just got in (in the appendix), other recent work is missing, and parts of the book "date" to several years ago. There is also some understandable lack of correlation; aureomycin is "rarely" bactericidal on page 17 and "bactericidal in therapeutic concentrations" on page 146, and *Ps. pyocyanea* on page 154 becomes *Ps. aeruginosa* on page 268. The bibliography runs to 41 pages of references, and much of the text seems a rather uncritical compilation. The reviewer is quoted as having stated that penicillin has reduced the mortality of actinomycosis from 80 to 20%, but the authors do not mention that this referred only to the thoracic form of the disease. (He also, by the omission of an initial, becomes two different people in the reference list.) There is no discussion of synergism and antagonism between antibiotics, and a lack of emphasis on the present inconstancy of bacterial sensitivity, particularly to the newer drugs. It is useless nowadays to prescribe by rule-of-thumb methods, particularly for chronic infections of the air passages or urinary tract. Laboratory tests are an essential guide to successful treatment, and a statement to the contrary made in the preface should have been amplified or explained in the section describing such tests.

It is inevitable that a book on what is now so vast a subject should be incomplete. Despite its omissions, this is a useful work of reference, as well as a generally reliable guide to therapy for the general practitioner, some specialists, and the veterinarian.

L. P. Garrod.

MEDICINE IN THE U.S.S.R.

La Médecine en U.R.S.S. By Dr. I. Lazarevitch. Preface by Dr. Jean Paraf. (Pp. 217. No price given.) Paris: Les Iles d'Or. 1953.

The great merit of this book is its objectivity. Written by a woman doctor of Russian birth but trained in France, it covers every aspect of Soviet medicine and is based entirely on recent official documents. It is impossible to describe here the details of organization, but the general impression left on the reader is that of a gigantic bureaucratic system planned on paper to the last detail but in practice failing in co-ordination and showing more regard for Communist ideology than for either patient or doctor. But there are some principles underlying the plan which indicate—to a Western mind at least—why it cannot work.

The first three chapters are devoted to an examination of what may be called the "Pavlov Creed," which by an official decree must dominate every branch of science, especially medicine. Many university professors have been deprived of their office and publicly denounced because they have shown independence of thought. It appears that certain theories propounded by Pavlov, who died in 1937, have been adopted by the Party as sacrosanct. The effect of this is that there is growing up a generation of medical teachers not free to question these theories, and of practitioners who are not allowed to know that in the view of scientists outside Russia no theory is sacrosanct. Moreover, in the *Meditsinski Rabotnik* of October, 1951 (official journal of the Ministry of Health), appears the following statement: "One of the essential duties of Soviet medicine is to establish the primacy of Russian savants in the different domains of medical science." Many examples are given in the book of the ridiculous claims that have been made in obedience to this decree. They must be seen to be believed.

There are some queer anomalies in the system. For example, the columns of the official medical journal seem to be open to severe criticism of the administration. Again, all doctors on qualification are immediately assigned to posts in various parts of the vast Soviet territory. There is no question of choice, and frequently the doctor on arrival finds the place very uncongenial and quite often no arrangements have been made for his accommodation. The result is that a considerable percentage of those assigned leave without permission. As there is no private practice in the Soviet it is difficult to understand what happens to these doctors. I can cordially recommend this book to anyone who wants to know what a really planned bureaucratic medical service is like.

Alfred Cox.

ENZYMES AND ANAPHYLAXIS

Enzymatic Concept of Anaphylaxis and Allergy. By Z. Z. Godlowski, M.D. (Cracow), Ph.D. (Edin.), M.R.C.P. (Edin.). Foreword by Professor A. Murray Drennan, M.D., F.R.C.P.Ed., F.R.S.Ed. (Pp. 120; 17 figures. 15s.) Edinburgh and London: E. and S. Livingstone. 1953.

Dr. Godlowski's theory is that, in the process of sensitization to an antigen, specific proteases are formed in certain cells. These enzymes will break down only the specific antigen, and break this down only to the stage of toxic proteoses.

The nature of the reagin, in the serum of allergic patients, that produces the Prausnitz-Küstner reaction is indeed a problem. Any theory should be considered, even when it postulates highly specific proteolytic enzymes which have not been shown to exist. But Dr. Godlowski also applies this theory to anaphylaxis, including passive anaphylaxis. The quantitative work of Kabat and his colleagues has shown that the sensitizing agent in passive anaphylaxis is almost certainly ordinary antibody.

In the second part of the book Dr. Godlowski maintains that certain eosinophilic cells are formed in the intestinal mucosa after absorption of foreign protein. The evidence for this is unconvincing and no explanation is given of the acquired affinity for eosin.

Some of the statements in the book are questionable: it is usually supposed that no breakdown of protein occurs in lysis, such as lysis of red blood cells by specific antiserum and complement (p. 5); there is no evidence to suggest that agglutination is due to the viscosity of newly formed protein molecules localized on the surface of bacteria or cells (p. 5); the definition of a haptene (p. 4) is wrong.

J. R. Marrack.

ABERRANT BEHAVIOUR

Encyclopedia of Aberrations. Edited by E. Podolsky, M.D. Foreword by Alexandra Adler, M.D. (Pp. 550. Price $10.) New York: Philosophical Publications. 1953.

The sketches of corsets and high-heeled shoes on the dust-cover and the title of the book suggest, and presumably are meant to suggest, that this is a comprehensive work on sexual deviations. This might be permissible on the hypothesis that the neuroses and all other mental disabilities are caused by disturbance in the development of the sexual function. But the validity of this hypothesis is not claimed, and sexual, social, religious, sensory, and other types of "aberrational behaviour" are described. The subjects chosen are dealt with by providing a simple definition or, when detailed consideration is called for, by reprinting an article previously published in a psychiatric journal. The selected articles are by representatives of the Freudian discipline of psycho-analysis.

The word "encyclopedia" is clearly inappropriate, as the terminology used by non-psycho-analysts is left out entirely. But even the psycho-analysts may complain about the omissions and so may "the intelligent laymen as well as the expert in interpersonal relations" whose requirements the editor has in mind. Transference, insomnia, sleep walking, trance states, impotence, vaginismus, and neurosis are forgotten. The number of neologisms is remarkable. Triakiadekaphobia (irrational fear of the number 13), hadephobia (the delusion that one has committed the unpardonable sin), and philonasm (obsessive interest in fads) are examples. The hotch-potch of good and bad, fact and theory, contained in this expensive volume has little value for the general physician or psychiatrist who possesses a reliable medical dictionary.

E. A. Bennet.

BRITISH MEDICAL JOURNAL
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CHRONIC GASTRIC ULCER

In their important review of the treatment of chronic gastric ulcer published this week in our opening pages, Dr. B. F. Swynnerton and Mr. N. C. Tanner stress the importance of considering gastric and duodenal ulcer separately. There is much to suggest that different factors are concerned in the causation of gastric and duodenal ulcer, and certainly their surgical treatment presents different problems. To provide an authoritative opinion in this field needs a particularly wide personal experience, and there are few centres where one individual has been responsible for nearly 500 patients with chronic gastric ulcer over seven years. This was possible in Mr. Tanner's gastric unit at St. James's Hospital.

Swynnerton and Tanner report a series of 498 patients with chronic gastric ulcer treated between 1940 and 1946. Of these, 262 had medical management after an active chronic ulcer had been diagnosed gastroscopically, while in the surgical series there were 254 patients; 18 patients appeared in both series. The posterior aspect of the upper part of the lesser curve was found to be the position of predilection for gastric ulcer in women, and ulcers in the pyloric antrum and canal constituted only 0.8%, as compared with 13.5% among men. In both sexes ulcers were found much more commonly on the posterior, rather than on the anterior, aspect of the lesser curve.

In the surgical series partial gastrectomy was the operation of choice initially with an antecolic Polya-Hofmeister, but this has now been replaced by the Billroth-I type, an operation which is considered a simpler and perhaps more physiological procedure. The surgical mortality for the whole series was 6.3%, but most of these deaths occurred during the first five months of intensive night bombing on London, and in the last 350 cases on which Mr. Tanner has performed partial gastrectomy there have been only four operative deaths, an incidence of just over 1%. This is a particularly fine record, for they include thirty-eight emergency operations for haematemesis.

A careful follow-up was organized and information obtained from all but fourteen patients—a remarkable achievement. Sixty-one patients in the medical and sixty-six in the surgical series had died since their initial stay in hospital; seven deaths were due to cancer of the stomach, nine to tuberculosis, and the remainder to a wide selection of diseases. Among the survivors in the medical series, 32% had undergone gastrectomy for troublesome relapses and only one-quarter had remained free from symptoms. Among the surgical cases, out of every ten patients still alive the results had been satisfactory in eight, moderately successful in one, and poor in one. Among the unsatisfactory cases there were often severe associated medical conditions, such as chronic bronchitis and angina pectoris. Analysis shows that at St. James's Hospital partial gastrectomy is the most satisfactory treatment of chronic gastric ulcer available at present; it is indicated except in those patients with a short history or with troublesome concurrent diseases. It is interesting to note that Swynnerton and Tanner are not convinced that partial gastrectomy increases the risk of pulmonary tuberculosis. The evidence from the literature clearly points in this direction, and further studies are needed.

The experience of this series does not lend any support to the view that simple gastric ulcer is liable to malignant degeneration. The view is increasingly held, at least in Britain, that most so-called "ulcer-cancers" are, in fact, "cancer-ulcers," the initial lesion being a malignant degeneration with a secondary ulceration. In a previous paper[1] Swynnerton and Truelove showed that the sites of these ulcers conform with the distribution of carcinoma of the stomach and not with that of gastric ulcer, the majority of them being prepyloric in situation. But many workers in the U.S.A. still insist that a gastric ulcer carries a 10% chance of malignancy. While a safe distinction cannot be made by examining the peritoneal surface during operation for perforation, in ordinary clinical practice the combination of careful radiology, aided when necessary by gastroscopy, can safely distinguish between the two. Radiologically, an ulcer cleanly projecting from the lesser curve above the angulus is virtually never malignant; the differential diagnosis arises when the ulcer is situated within the line of the lesser curve. Size is of no real significance, small lesions often being among the most suspicious.

In reviewing the cases of gastric carcinoma which had developed during the follow-up, Swynnerton and Tanner did not consider that there was any evidence that they had begun as ulcer-cancers. They con-

[1] Swynnerton, B. F., and Truelove, S. C., *British Medical Journal*, 1951, 2, 1243.
[2] Cain, J. C., *et al.*, *J. Amer. med. Ass.*, 1952, **150**, 781.

cluded that, if there is an association between gastric ulcer and gastric carcinoma, it is because both lesions are apt to develop in the same type of degenerative mucosa. There is certainly some evidence that patients who have suffered from gastric ulcer are more liable subsequently to develop gastric carcinoma. Cain and his colleagues,[3] in a series of 414 patients followed up for five years after medical treatment of gastric ulcer, found that 4.9% had developed gastric carcinoma, an incidence which is many times greater than among the general population. From the histories it seemed highly probable that many of the carcinomas were unrelated to the original ulcer. Again, the removal of the ulcer by partial gastrectomy does not confer immunity from subsequent malignant disease. An interesting feature of the American series is that the proportion of completely successful cases treated medically, 20%, is almost identical with Swynnerton and Tanner's figure of 23.6%. The proportion of medically treated cases subsequently coming to surgery was also remarkably similar in the two series, being 32% and 33.8%, east and west of the Atlantic.

In spite of these impressive figures it is doubtful if it would yet be generally accepted that gastric ulcer is essentially a disease that must have surgical treatment, for there are many patients in whom gastric ulceration is an occasional episode. This is particularly true of cases of acute gastric ulcer, in which the prognosis, especially in women, is far better than with chronic ulcers. Although a high proportion of patients treated medically may have subsequent relapses, yet a high proportion of their follow-up years are entirely free from symptoms. For a persistently unhealed ulcer there can be no doubt at all that surgery is the correct treatment, but most clinicians would still prefer to give every patient with gastric ulcer an initial chance to respond to medical treatment, and indeed it is surprising how many may heal while on a hospital waiting-list. It is not uncommon for an ulcer to develop after physical or mental stress, particularly if associated with under-nutrition or some other disease, such as bronchitis. In these circumstances there is often much which can be done by the physician together with the almoner in improving the patient's circumstances and getting him readjusted to his individual problems.

There are many diseases in which the best results in any series of cases are achieved only by close co-operation between physician and surgeon. This is especially true of hypertension, thyrotoxicosis, ulcerative colitis, and peptic ulcer. In these conditions the decision to operate should be agreed on by both the physician and the surgeon. The value of such co-operation is also to be seen when physician and surgeon collaborate in the analysis and publication of the results of treatment, as they have done in this series.

STATISTICS AND MEDICINE

The vital statistics of this country—and indeed of the world—owe much to the genius of Dr. William Farr, medical statistician at the General Register Office from 1839 to 1879, and it is fitting that in Somerset House has been held this week the first World Health Organization Conference of National Committees on Vital and Health Statistics, under the presidency of Sir Russell Brain. National committees on vital and health statistics are of recent origin. They were first suggested in 1948 at the Sixth Decennial Revision Conference of the International List of Diseases and Causes of Death, when it was recommended that governments establish such committees for the purpose of co-ordinating statistical activities within their own countries and to serve as links between national medical-statistical institutions and the World Health Organization. These national committees would have the function of studying broadly the problem of producing satisfactory national and international statistics in the field of health. National committees have now been established in some twenty-five countries, and a number of others, including Britain, though lacking a formal national committee, have co-ordinating bodies and arrangements that amount to much the same thing.

The conference, attended by delegates from some thirty countries and international organizations, and with the United Kingdom strongly represented, has been discussing the structure and functions of these national committees. In addition it has devoted time to questions such as the types of health statistics essential to countries at different stages of development, how those statistics are to be obtained, and what part W.H.O. ought to play in this connexion. Other topics discussed have been the extent to which health statistics might be obtained by sampling rather than by more traditional methods, and the national implementation of international regulations on vital and health statistics, particularly in relation to death certification and the classification of diseases.

The conference has been dealing with topics somewhat remote from clinical medicine. Indeed, it is a criticism sometimes made that vital statistics in general provide little of interest or of direct value to the majority of medical practitioners. There is

no doubt an element of truth in this, for the general practitioner and the hospital consultant, going about their daily affairs, find little need to delve deeply into national and local vital statistics. Statistics are unpopular but necessary: they are troublesome to collect and compile, some of them are difficult to understand without the requisite training, and they have a habit either of failing to provide a convincing answer to the question asked or, worst sin of all, of contradicting preconceived ideas and long-held beliefs. Moreover, not even the most enthusiastic advocate of vital statistics would claim that statistical tables of births, marriages, and deaths make exciting reading. Yet vital statistics are not a mere academic luxury. They have a very real practical value in almost every field of medicine. A knowledge of prevalent infectious diseases and causes of death, for instance, is useful to most doctors, and may be gained from a scrutiny of the vital statistics section in this *Journal* each week.

One of the topics discussed at this W.H.O. conference was how to secure a wide appreciation of the value of vital statistics and how to obtain the co-operation of those who could supply needed data. Perhaps the answer lies not only in giving medical students instruction in statistics, their sources, their value, and their limitations—as suggested by Professor Bradford Hill at the recent First World Conference on Medical Education[1]—but also in trying to present vital statistics in a way that will appeal not only to statisticians but also to medical men, who are after all the people most concerned with the true import of those statistics. We are frequently reminded that, when undertaking a piece of research likely to require statistical analysis, the medical worker should consult a statistician at the beginning rather than at the end if effective collaboration is to be achieved. But the opposite also holds, and in the preparation and presentation of vital statistics, and indeed also in presenting the results of any investigation requiring statistical analysis, the statistician must likewise in turn seek medical advice, and ensure that what he is offering is of direct value to medical people and can be readily understood by them.

We have been fortunate in this country that our national vital statistics have all along been prepared under the supervision of men with a medical as well as a statistical training. The Statistics Division of W.H.O. also enjoys this advantage, but many national statistical offices are less fortunate, and have to rely on statisticians without medical qualifications. Unless these statisticians obtain the best possible medical advice, their contribution will not properly fulfil the requirements of the medical profession whom they hope to serve. Vital statistics are essentially a medical rather than a statistical exercise; the medical profession supplies much of the basic data, and is the principal consumer of the resultant statistics: its tastes and needs must be consulted in deciding what statistics ought to be prepared and how they should be presented.

[1] *British Medical Journal*, 1953, **2**, 487.

HEARTBURN IN PREGNANCY

The pregnant woman and her doctor do not always see things in the same perspective, and what the doctor considers a minor discomfort his patient may regard as a major affliction. This is particularly true of heartburn, which in some degree harasses two out of every three pregnant women. Formerly this symptom was blamed on hyperchlorhydria, but alkalis give inconstant relief, and in most pregnant women there is a notable reduction in the free and total gastric acidity, particularly during the second trimester.[1] Latterly, therefore, heartburn has been treated with small doses of dilute hydrochloric acid, but again with variable success. Williams[2] reported that in addition to a reduction in acidity there is a diminution of gastric motility in pregnancy, and it has been suggested that this, together with a neuromuscular disturbance at the cardio-oesophageal junction, allows regurgitation into the oesophagus. Williams therefore advocates the use of prostigmin to stimulate gastric motility and asserts that a single injection of 0.5 mg. may relieve heartburn for several days.

In a paper on page 864 of the *Journal* this week Dr. W. A. W. Dutton and Dr. H. J. Bland draw attention to herniation of part of the stomach through the oesophageal hiatus of the diaphragm as a cause of heartburn in pregnancy. A hiatus hernia may be asymptomatic, but they diagnosed nine cases in pregnant women within three years. The chief clinical features are heartburn, epigastric discomfort, and vomiting. The last may be persistent and severe, particularly during labour, and may culminate in a haematemesis, especially if peptic ulceration of the oesophagus supervenes, while the onset of dysphagia may betoken the formation of an oesophageal stricture. Diagnostic features are that all these symptoms are aggravated by lying down and by stooping forward, and become more severe in the last months of pregnancy. It might be thought possible that the bearing-down strain of the second stage of labour might precipitate a hiatus hernia, but four of the nine women in this series were primigravid. Moreover, all reports agree not only that the symptoms abate after delivery but also that the hernia may become much smaller or even be no longer demonstrable radiographically a few months after the end of the pregnancy. Nevertheless it should be borne in mind that hiatus hernia is commoner in later life, and it may be that a long-term follow-up of cases diagnosed during pregnancy might reveal that the condition recurs when the woman is older and fatter.

[1] Strauss, M. B., and Castle, W. B., *Amer. J. med. Sci.*, 1933, **185**, 539.
[2] Williams, N. H., *Amer. J. Obstet. Gynec.*, 1941, **42**, 814.
[3] Osborne, W. W., and Foster, C. D., ibid., 1953, **66**, 682.

Heartburn in pregnancy may therefore be of some importance, and certainly the more severe and persistent cases, especially if associated with vomiting or dysphagia, deserve radiographic investigation. Not only will a certain number of cases of hiatus hernia be discovered thereby, but there will also be brought to light the uncommon case of more extensive diaphragmatic hernia, which carries a high mortality because it is rarely diagnosed before the onset of obstruction or strangulation.[3]

FEEDING CHILDREN WITHOUT MILK

In the western world mothers who are either unable or unwilling to feed their babies at the breast can rely on liberal supplies of efficient substitutes made from cows' milk. After the period normally occupied by lactation, it is considered sound policy for the child to be given large quantities of milk right up to adolescence. In many Oriental and tropical countries, however, cows' milk in any form is either scarce or absent. In order to bridge the gap between the end of breast-feeding and the development of digestive powers strong enough to cope with the normal local diet, vegetable substitutes for cows' milk are urgently needed. Countless lives are being lost because in many areas no suitable alternative is available.

The value of vegetable foodstuffs is generally considered to be limited by the quality of their proteins. Although human milk is not particularly rich in total protein, its contribution of some of the amino-acids—particularly tryptophan, cystine, and valine—is difficult to equal from vegetable sources. As a general principle mixtures of vegetable proteins are better than single proteins, because their strengths and weaknesses in the individual amino-acids tend to cancel each other out. In 1944 Italian workers in Rome found that foods made from mixtures of malted cereals were useful for infant feeding at a time of acute food shortage. Improved foods of this type, with their protein contents increased by the inclusion of soya flour, were brought to the notice of Dr. Harriette Chick, who confirmed their nutritional value in experiments with rats.[1]

Further trials in which cereal mixtures were given to children in German orphanages and schools have now been reported by Dr. R. F. A. Dean.[2] Various combinations of barley, soya, and wheat or maize, sometimes fortified with milk, were given as part of the diet to about 25 newborn babies and to over 700 children of ages up to 11 years. The results seem hopeful, although it is clear that a perfect vegetable substitute for milk has still to be devised. For infants up to 1 year old about half of the milk in the diet could be replaced by some of the mixtures. For children from 1 to 2 years old at least two mixtures seemed nearly perfect substitutes for all the milk in the diet. For children aged between 2 and 11 years almost all the mixtures gave good results, but for optimum growth a small allowance of milk was necessary.

Difficulties in interpreting the results arose from illnesses unconnected with the diet and from variations in the methods of manufacturing the cereal mixtures. Thus soya flour contains a trypsin inhibitor, which had to be removed or destroyed by heating before the foods could be efficiently digested. Some mixtures caused diarrhoea, or were regurgitated by the younger children. Apart from the trypsin inhibitor, suspicion falls on carbohydrate rather than on protein as the component affected by the mode of manufacture. For the present, therefore, it seems that theoretical considerations of protein requirements must take second place to the rule-of-thumb methods of food manufacturers in deciding whether human babies can subsist on cereal mixtures. More intensive investigation of the chemical changes occurring during the processing of milk substitutes is urgently needed.

1 Chick, H., and Slack, E. B., *Lancet*, 1946, **2**, 601.
2 Dean, R. F. A., *Plant Proteins in Child Feeding*, Medical Research Council; Special Report Series No. 279. 1953. London: H.M.S.O.

LIVER BIOPSY

The practice of liver biopsy as a diagnostic procedure has increased in recent years, chiefly because of the introduction of new instruments most of which function on the principle of the trocar and cannula.[1-4] In assessing the value of the procedure two aspects of it have to be borne in mind. First is the question of its dangers and complications, which have figured largely in the literature produced by both its advocates and its antagonists, while secondly the adequacy of the biopsy material obtained and the accuracy of its interpretation deserve attention.

The dangers and complications may be grouped under the headings of haemorrhage, infection somewhere along the needle track, and, more rarely, bile peritonitis. Occasional deaths have been recorded.[5] They are most commonly due to haemorrhage caused by tearing of the capsule or by bleeding into the liver parenchyma in patients suffering from diseases causing prolonged bleeding times. For this reason a prolonged bleeding or clotting time or a prothrombin time of less than 50% of normal should be contraindications to liver biopsy. Patients should be watched carefully for 24 hours after liver biopsy, and cross-matched fresh blood should be held ready for an emergency.

The technique chosen may increase or diminish the dangers from haemorrhage. In some methods the approach is intercostal and entails fixation of the needle, its rapid introduction and withdrawal, and cessation of respiration during the manipulation. When the liver is palpable below the costal margin an abdominal approach can be used, which is clearly safer in that the needle can more freely enter the liver.

The adequacy of the biopsy material obtained largely depends on the type of needle used, and with modern instruments cores of tissue some 2–3 mm. in diameter measuring 2–3 cm. in length can be obtained. The histological examination of a piece of tissue of this size is technically easy, and after suitable fixation affords opportunities for studying liver material in a fresh state, opening up a new field of observation which has obvious advantages over post-mortem examination. As a

1 Silverman, I., *Amer. J. Surg.*, 1938, **40**, 671.
2 Gillman, T., and Gillman, J., *S. Afr. med. J.*, 1945, **10**, 53.
3 Sherlock, S., *Lancet*, 1945, **2**, 397.
4 Sborov, V. M., *J. Lab. clin. Med.*, 1950, **36**, 773.
5 Davis, W. D., *et al.*, *Amer. J. med. Sci.*, 1946, **212**, 449.
6 Harvier, P., *et al.*, *Presse méd.* 1953, **61**, 423.
7 Sborov, V. M., and Blumberg, J. M., *J. Amer. med. Ass.*, 1953, **151**, 1070.

research tool, therefore, in spite of its possible dangers, liver biopsy is useful. But the question of its value in practical diagnosis is a different one. Where a minute fragment of a large organ is being examined, the morbid changes must be diffuse and uniform in distribution if the appearances are to be representative. Again, if the pathological process is focal or if it varies considerably from one point to another, then interpretations may be inaccurate or even confusing. Thus in cases of toxic or virus hepatitis, or diffuse hepatic fatty degeneration or fibrosis, reasonably accurate results may be expected and interesting observations may be made. In cases of malignant infiltration, abscess formation, or any other focal abnormality the diseased area may be completely overlooked. It is therefore with the former group of conditions that most reports on the methods used are concerned.[6,7]

At present it may be said that considerably more experience is required before a final judgment can be passed on liver biopsy. It is probably best confined to teams where both clinician and pathologist are experienced in its use and where proper precautions will be taken. In those circumstances it is an informative research procedure and, as a diagnostic test, useful chiefly in those diseases which produce diffuse changes in the liver.

BULBAR POLIOMYELITIS AND TONSILLECTOMY

The now well-known association between bulbar poliomyelitis and tonsillectomy, based chiefly on American experience, has been the subject of comment before in these columns, and there is general agreement that tonsillectomy should be postponed when possible until the risk of an autumn recrudescence in the outbreak is past. Although the evidence is conflicting on whether tonsillectomy predisposes to the contraction of poliomyelitis, there seems to be no doubt that, if the disease is contracted, it is more likely to be of the bulbar type. The risk of bulbar paralysis is considered to be up to sixteen times greater in patients who have had their tonsils removed. Some new work on this subject now comes from Australia.

Southcott[1] has made a detailed analysis of the data from the 1947–8 epidemic in South Australia; 103 paralytic cases were recorded, and of these 56 had had tonsillectomy. In this outbreak 40 cases of bulbar paralysis occurred, 35 in tonsillectomized patients, 4 in non-tonsillectomized, and 1 in a patient whose history of tonsillectomy was unknown. Only one patient had had recent tonsillectomy—a 15-day interval between operation and disease. Sixteen of the 35 patients had intervals of five or more years between operation and contraction of poliomyelitis. Although the number of patients with long intervals was small, it does seem that the effect of tonsillectomy lasts at least ten years. The age distribution of the bulbar and non-bulbar paralytic cases differed considerably. No child under 5 contracted bulbar paralysis, while 11 had non-bulbar paralysis; at ages 5–14 there were 28 cases of bulbar and 19 of non-bulbar paralysis. Sixteen of the 40 cases of bulbar paralysis and 4 of the 63 cases of non-bulbar paralysis were fatal. No evidence could be obtained from the data to show that any relationship existed between tonsillectomy and non-bulbar paralysis or non-paralytic poliomyelitis.

[1] Southcott, R. V., *Med. J. Aust.*, 1953, **2**, 281.

OSTEOCHONDRITIS DISSECANS

Although osteochondritis dissecans is commonly regarded as a complaint of the young adult, it usually begins during adolescence. Most of the patients are males. In two series of cases of osteochondritis of the knee[1] and elbow,[2] reported by Green and Banks and by Roberts and Hughes respectively, 55 out of a total of 65 patients were boys, and all the patients were under 15.

The condition consists in an avascular necrosis of an area of bone lying beneath articular cartilage. Its cause is not known with certainty. The onset during adolescence, the many patients in whom several joints are involved, and the associated epiphysial prematurity sometimes seen suggest that an endocrine disturbance may start up the condition. Of possible local causes trauma is thought the most likely, for there is a history of injury in many cases, most of the patients are males, and when the elbow-joint is affected it is usually the right.

An attack of locking during adult life may be the first indication of osteochondritis dissecans, particularly in the elbow-joint. In its early stages the condition must often escape recognition, for the physical signs and symptoms are easily overlooked. Indeed, in many cases there are no significant physical signs. Clear radiographs, sometimes giving special views, are essential.

Green and Banks have shown that the best treatment of osteochondritis dissecans of the knee in children is conservative. In their series this consisted in immobilization in a plaster-of-Paris cylinder or in a weight-relieving calliper for four to seven months. Full weight-bearing was permitted in plaster, but the joint was fixed in a position which prevented pressure on the osteochondritic area.

Healing occurs through revascularization by creeping substitution of avascular bone, even when there is evidence of sequestrum formation. The articular cartilage, which remains healthy during the early stages, must be protected from damage, for if it becomes fissured it may separate from the underlying bone and form a loose body. In the series reported by Green and Banks 25 out of 36 joints were treated conservatively; 18 patients were followed up for one year, and in 17 the results were excellent.

We record with great regret the death at Cardiff on October 13 of Sir Ewen Maclean, emeritus professor of obstetrics and gynaecology at the Welsh National School of Medicine. He was Chairman of the Representative Body from 1910 to 1912 and President of the B.M.A. in 1928.

[1] *J. Bone Jt Surg.*, 1953, **35-A**, 26.
[2] Ibid., 1950, **32-B**, 348.

CLINICAL PATHOLOGY IN GENERAL PRACTICE

DISPATCH OF SPECIMENS

BY

MARTIN HYNES, M.D., M.R.C.P.

Clinical Pathologist, Royal Northern Group of Hospitals

The clinical pathologist is a bridge between the laboratory and the clinician, and can function only if he is as familiar with the patient's state as with his laboratory investigations. It is possible to churn out mechanical reports on swabs from unknown sources and blood from unknown diseases, but this is technicians' work, not pathologists'. Only when the pathologist is informed of the clinical aspects of the case can he select the most appropriate means of investigating the material sent to him, and interpret his findings to suggest a diagnosis or further investigations. The minimum information sent with every specimen should be:

The patient's name, age, and sex.

The source of the material, and when it was taken.

The investigations required.

The essentials of the clinical history, including details of any chemotherapy.

The probable diagnosis.

When the case is difficult the pathologist and clinician should at least consult by telephone, and should preferably meet at the bedside.

It is easy to forget to label a specimen, and thus to destroy all certainty that the pathological report truly belongs to the patient in question. The patient's name and the date and time of taking the specimen are essential, but it is wise to add to the label some other details, such as the patient's age or the doctor's name, in case, as not infrequently happens in busy laboratories, two patients of the same or very similar names are being investigated at the same time.

FIG. 1.—Bottle for liquid specimen, with wooden container.

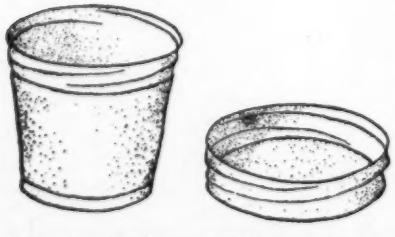


FIG. 2.—Waxed cardboard container for faeces or sputum.

Containers

Only four types of container, some throat swabs, and glass slides are needed for the great majority of specimens for pathological examination. All can usually be borrowed from the laboratory that will perform the investigations, for it is not reasonable to expect the general practitioner to clean and sterilize his own glassware. But, if he must, glass containers are most conveniently sterilized at home by using the ordinary domestic pressure-cooker as an autoclave: 20 or 30 minutes' cooking at full pressure is adequate.

Liquid specimens should be placed in sterile wide-mouthed screw-capped bottles (Fig. 1); corks too readily come out in transit. Three types are required:

1. Plain sterile 1-oz. (28-ml.) bottles to contain blood for serum, urine, and other fluids for culture, etc. Wash the bottles well but do not use detergents, which grossly interfere with the Wassermann reaction and some other tests. Dry thoroughly and sterilize in the pressure-cooker with the caps lightly screwed on the bottles.

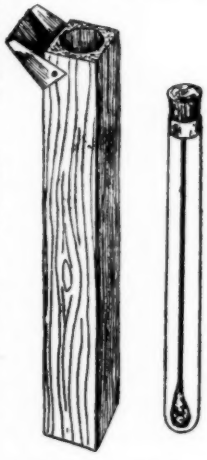


FIG. 3.—Throat swab tube, with wooden container.

2. Oxalate bottles, for most biochemical estimations on blood. Add a knife-point of sodium or potassium oxalate to a plain bottle prepared as above.

3. Wintrobe bottles for blood counts and E.S.R. (sedimentation rate) determinations. In a sterile ¼-oz. (7-ml.) screw-capped bottle, prepared as above, place 0.2 ml. of a solution containing 2 g. potassium oxalate and 3 g. ammonium oxalate in 100 ml.; evaporate to dryness in a warm but not hot place. A bottle thus prepared is for 5 ml. blood.

Faeces and *sputum* are best sent to the laboratory in waxed cardboard containers (Fig. 2) which can be burnt after use. These containers need not be sterilized.

Throat swabs can be purchased prepared ready for use, together with suitable containers for the post (Fig. 3). They should be sterilized before use (unless obtained from a laboratory) by pressure-cooking for 30 minutes.

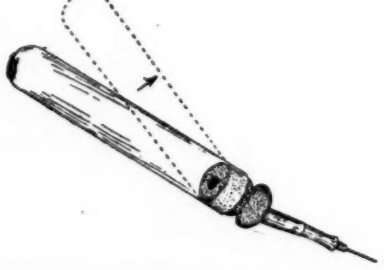


FIG. 4.—Behring venule. The dotted line shows the position when the vacuum is released.

Glass slides can be used straight from their box. If they are washed they should be dried with a freshly laundered grease-free linen cloth. There is no advantage in storing them in spirit.

Less frequently used containers, such as bottles of broth for blood culture, fluoride tubes for blood-sugar estimations, and jars of formalin solution for histological material, will certainly be supplied by the laboratory when they are required.

The *Behring venule** (Fig. 4) conveniently replaces both syringe and container. It comprises a vacuum tube to which is attached a sterile needle protected by a sealed glass covering. When this covering is removed the vacuum is preserved by a simple valvular arrangement until, when the needle is

* Obtainable from Bayer Products Ltd., Africa House, Kingsway, London, W.C.2.

in the vein, the needle is flexed on the tube. When the valve is thus opened blood is drawn into the container. Venules are prepared to correspond to the plain bottles, oxalate bottles, and Wintrobe bottles described above, as well as one containing glucose broth for blood culture. Containers suitable for postal delivery can be obtained with the venules.

Specimens for Bacteriological Examination

The proper selection of material for bacteriological examination often demands a little thought. For example, pathogens are always present in the advancing edge of a lesion, whereas they may have died out in the necrotic centre.

TABLE I.—*The Viability of Common Pathogenic Organisms on Swabs, etc.*

VERY RESISTANT (Survive for days or weeks)
- Staphylococci
- Streptococci
- *Bact. friedländeri*
- Diphtheria bacillus
- Tubercle bacillus
- *Actinomyces bovis*
- Anthrax bacillus
- Clostridia (tetanus, gas-gangrene, etc.)
- Viruses
- Rickettsia (typhus, etc.)
- Fungi
- *E. histolytica* cysts

VERY DELICATE (Survive at most a few hours when dry)
- Meningococcus
- Gonococcus
- *T. pallidum* (syphilis)
- *E. histolytica*, vegetative forms
- Trichomonas

INTERMEDIATE (Survive two days or more)
- Pneumococci
- *Bact. coli*
- Salmonellae (typhoid and food-poisoning)
- Shigellae (dysentery)
- Haemophilus (whooping-cough, *H. influenzae*)
- Brucella (abortus and Malta fever)

With a few exceptions (Table I) pathogenic bacteria readily survive the normal time of transit to the laboratory. Drying and exposure to sunlight are the most deleterious factors; chilling does little or no harm. Even gonococci and *Trichomonas vaginalis* survive for two days or more in Stuart's transport medium, which keeps them moist while suspending their animation by lack of oxygen. A further disadvantage of delay is that saprophytic bacteria may multiply in the specimen at room temperature and come to outnumber the pathogens. The pathologist can often counteract this process by the use of selective media, but it is better to avoid it. Finally, it is necessary to remember that sulphonamides and antibiotics may suppress causative bacteria without curing the patient. If specimens are not taken before starting treatment diagnosis may be greatly delayed or even made impossible. If specific therapy has already been started, this fact should be noted.

Blood Culture.—Many bacteria are rapidly killed by the patient's undiluted blood (the typhoid–paratyphoid group are an exception). Blood for culture should therefore be diluted immediately in bottles of the appropriate medium, using about 7 ml. blood to 50 ml. medium. Ordinary broth suffices for most bacteria, but *Str. viridans* prefers glucose broth. If rarer infections are suspected (for example, meningococcal septicaemia or brucellosis) the pathologist should be asked to supply the most appropriate medium. Special media, containing *p*-aminobenzoic acid, are also required if the patient is receiving sulphonamide therapy.

Blood culture demands a careful but not an elaborate technique. The patient's arm need be cleaned only as for any venepuncture, and sterile towels are not needed. The most common sources of contamination are imperfect sterilization or careless handling of the syringe and needle, repeated attempts to enter a vein, a leaky syringe, or careless exposure of the broth while the blood is added.

Faeces.—Faeces are best carried to the laboratory in a waxed cardboard container, which can be burned. A rectal swab is as efficient as faeces for the bacteriological diagnosis of bacillary dysentery, and may save valuable time in the diagnosis of enteric fever when faeces are not available. Helminth ova are generally found in faeces, but it must be remembered that threadworm ova are deposited outside the bowel on the perianal skin. They readily adhere to a piece of "cellophane" rubbed gently over the skin; the cellophane is sent to the laboratory in a screw-capped bottle.

Pus.—A throat swab of the pus, with a smear if more than an hour or two's delay is likely, is sufficient for the diagnosis of pyogenic infections. If tuberculosis or actinomycosis is suspected, 5 or 10 ml. of the pus should be sent to the laboratory in a sterile 1-oz. (28-ml.) screw-capped bottle.

Hairs.—The infected stubs of hair in ringworm infection need to be carefully chosen. Lengths of hair broken or snipped off are quite useless, for the infection is in the hair root. It is best to send the stubs to the laboratory in a piece of white paper folded in the manner of a chemist's powder packet.

Scales.—Scales of skin may be sent to the laboratory in a paper packet like hairs (above) or may be enclosed between glass slides held together by rubber bands. The scales must be chosen from the advancing edge of the lesion, not from the centre.

Pleural Fluid (and other serous fluids such as ovarian or ascitic fluid) should be sent to the laboratory in a sterile 1-oz. (28-ml.) screw-capped bottle. A part should also be sent in an oxalate bottle for cytological examination. If *malignant cells* are to be looked for the fluid should be diluted with at least an equal volume of 10% formalin solution (see next page).

Sputum is best carried to the laboratory in a waxed cardboard container, which can be burned. An early-morning specimen is best, but the patient should be reminded that saliva is useless. Sputum to be examined for carcinoma cells should be sent to the laboratory as promptly as possible. Formalin or other fixatives are deleterious.

Swabs.—The ordinary throat swab is a convenient means of sending a variety of materials to the laboratory for bacteriological examination. Most bacteria survive well on the swab (see Table I), but good smears cannot be made if it has dried. A smear should therefore be made on a slide (sterilized in the flame and cooled) if more than a few hours' delay is likely. Pernasal swabs, passed through the anterior nares along the floor of the nose to the nasopharynx, are at least as efficient as post-nasal swabs, as well as being less troublesome to take.

Urine.—A mid-stream specimen of urine from the male suffices for bacteriological examination, but a catheter specimen must be obtained from females if culture is required. In doubtful cases the women need not be subjected to the discomfort of catheterization until microscopy of an ordinary specimen of urine has revealed the presence of pus. Examination for tubercle bacilli should always be made on an early-morning specimen of urine. A 24-hour specimen is often desirable if it can be carried to the laboratory. An early-morning specimen is also required for pregnancy tests.

Haematology and Serology

Blood in ordinary oxalate bottles is of little use for any haematological examination except a simple haemoglobin estimation. Blood for counts and E.S.R. determinations should be placed in Wintrobe bottles. The amount of anticoagulant used is near the minimum, so the blood and oxalate must be thoroughly mixed by repeated inversion of the bottle at intervals for two or three minutes. Even the Wintrobe oxalate mixture soon distorts leucocytes; therefore two or three blood smears should be sent with the

specimen. It is important to remember that a too-thin blood smear always gives some information whereas one that is too thick may be completely useless. A smear is generally too thick if it does not peter out before the end of the slide. Blood in Wintrobe bottles can be used for haemoglobin determinations and red-cell counts for at least two days after taking the blood, but the leucocyte count begins to fall after a few hours and may be misleadingly low after 24 hours. The sedimentation rate is also spuriously low at periods greater than three hours after taking the blood.

Blood Grouping (including the Rh factor) is best done on blood allowed to clot in a plain bottle. The specimen must arrive at the laboratory within 24 hours of withdrawal if rhesus antibodies are to be sought for.

Blood for Serological Tests.—Blood for the Wassermann reaction, Widal reaction, and other serological tests should be allowed to clot in a sterile screw-capped bottle. Contamination of the blood should be avoided. Haemolysis, whether from water or spirit in the syringe or from bacterial contamination, vitiates the W.R. and many other tests. Blood from infants for the W.R., etc., is simply obtained by pricking the well-warmed heel and allowing the blood to flow into a Wright's capsule (Fig. 5).

Fig. 5.—Wright's capsule.

Biochemical Estimations

The majority of biochemical estimations on blood can be performed with either serum or plasma from oxalated blood. Serum is generally preferred to plasma, and is essential for a few estimations (Table II). Wintrobe bottles should not be used for blood for biochemical determinations; in particular, blood from them cannot be used for urea determinations. Most biochemical determinations are valid if done within a day or two of the withdrawal of the blood; the exceptions are indicated in Table II. The *blood sugar* can be satisfactorily estimated after 36 hours if fluoride is used instead of oxalate.

Table II.—*Containers for Blood for Biochemical Estimations (Minimum quantity of blood 1 ml. except where indicated)*

Plain or Oxalate Bottles
Bilirubin (minimum 2.5 ml. blood)
*Chlorides
Cholesterol
*CO_2-combining power (minimum 5 ml. blood)
Non-protein nitrogen
Phosphatase (acid or alkaline)
*Phosphates (minimum 2.5 ml. of blood)
Proteins (minimum 2 ml. of blood)
*Sugar
Urea
Uric acid
Plain Bottles Only
Serum calcium (minimum 5 ml. of blood)
*Serum potassium
*Serum sodium
Thymol turbidity

* Estimations marked thus must be made within two hours of withdrawing the blood.

Faeces for the occult blood test should be sent to the laboratory in the same way as bacteriological specimens. Other biochemical tests on faeces, urine, etc., are rarely needed in general practice. If any should be required, the advice of the laboratory should be sought before sending the specimen.

Histological Examinations

Material for histological examination must be sent to the laboratory in a fixative solution, otherwise autolysis may make interpretation of the sections impossible. The most generally used fluid is a 10% aqueous solution of commercial formalin. Screw-capped jars of all appropriate sizes can be obtained. Larger pieces of material should be wrapped in gauze before being placed in their container.

Postal Regulations

Every effort should be made to send pathological specimens to the laboratory by hand, thus eliminating delay and minimizing the possibility of loss or breakage. The Post Office regulations permit pathological specimens to be sent by *letter* post to a recognized medical laboratory or a qualified medical practitioner. Sending such specimens by parcel post is absolutely prohibited. The material must be placed in a securely closed container, which must itself be enclosed in a strong wooden or metal box in such a way that it cannot shift about, and with sufficient sawdust, cotton-wool, or other absorbent packing to prevent any possible leakage from the parcel in the event of damage to the container. The packet must be conspicuously marked "Fragile with Care" and bear the words "Pathological Specimen." Figs. 1 and 3 illustrate examples of containers and outer boxes that comply with the regulations.

Next Article on Clinical Pathology.—"Venepuncture," by Dr. I. A. B. Cathie.

Refresher Course Book.—The first collection of articles in the Refresher Course for General Practitioners (fully revised) are available as a book containing 55 chapters, price 25s. Copies can be obtained either direct from the Publishing Manager, B.M.A. House, Tavistock Square, London, W.C.1, or from booksellers.

PREVENTION OF TRANSFUSION ACCIDENTS

CONSULTING PATHOLOGISTS' GROUP CONFERENCE

The Consulting Pathologists' Group Conference was held at B.M.A. House on September 30. Professor D. F. Cappell was voted to the chair. Dr. J. G. Greenfield, Chairman of the Group Committee, presented its report, which was approved.

Dr. George Discombe opened a discussion on blood transfusion, its dangers, and the precautions which should be taken. The form of the problem was determined to a large extent by one's clinical colleagues. It sometimes happened that blood had to be provided in a hurry, perhaps several times in a day. Since 1946 in his department they had transfused about 14,000 bottles. Some 6,000 patients had received blood, and among those 6,000 only about 6% had developed some sort of reaction. Ten recognizable haemolytic reactions were found. In two of these no cause was discovered; eight were ABO incompatibles. He had been brought up to believe that in many cases of incompatibility death ensued, but in the cases of incompatible transfusion experienced in his department there had been no deaths.

For a long time they had used capillary blood for grouping and found it satisfactory. But their technicians had been instructed to look for the slightest deviation from the normal, and if this happened they immediately sought advice and help. Mistakes were analysed, and it was found that three mistakes were due to hurry, two were due to blunders made by an inadequately trained member of the laboratory staff who gave out blood to the wrong patient, and one was due to a nurse who had not read a label on the bottle. Laboratory administration had since been tightened up so that fewer people handled the blood, and recently a unit-recording system had been introduced. Thus mistakes were obviated whereby in one case two women with the same surname, one called Margaret and the other

Peggy, were confused. If a guiding statement was needed one required a serologist, regional transfusion officers, and at least two hospital transfusion officers to put their heads together and produce an agreed document. "I believe," he said, "that the main reason why I have not spent most of my time at the coroner's court is because I have kept a very close watch on my transfusion problems, even to taking over the cross-matching and doing it all myself, and also being available to my technicians for any help I can give them."

It was comforting to be given a set of rules by which to conduct one's practice, but he did not think one could ever frame rules in such a way as to make them perfect, and if they stuck to rules rigidly "sooner or later somebody will die in consequence." The choice had to be left to the individual consultant.

Too Much Blood Ordered

Dr. J. F. HEGGIE said that many of his views had been expressed in articles in the *British Medical Journal*. The pathologist had been given the responsibility of the blood banks as regards matching, supply, and delivery to the wards, as well as collaboration with his colleagues to make sure that the number of patients placed at risk was kept to the minimum. Often blood transfusion had been prescribed by quite junior persons when it was not necessary, and in view of some accidents they had been asked to look into the matter. It was found that the situation varied enormously in different regions. Generally, however, there was a tendency to order too much blood, and so a memorandum had been formulated instructing their colleagues on the risks which they were running and the greater serological care required in cross-matching blood in people who were having to be transfused for the second or third time. They had also asked that the element of urgency be so far as possible avoided.

The hospital officers concerned were told of the substitutes that could be used immediately; also that healthy young women suffering from abortion did not in general lose as much blood as might warrant transfusion. They were also reminded that the supply of blood was limited. The methods of grouping and matching in different laboratories had been reviewed, and the advisers to the Ministry were called in and a list of what were considered to be reasonable precautions and techniques was approved. If a hospital were large enough a resident pathologist should be employed. But there were many places in his region where the hospitals were small, and in those areas it had been ensured that a senior technician or a technician suitably experienced in blood grouping and matching should be available for emergencies. All this ensured that the standard of technique was good and that it was not a matter of having a house officer engaged who had merely been shown how to group and cross-match for a week or so.

In hospital administration the rules with regard to taking blood from the bank had been made more rigid. In his own hospital it had been arranged that no nurse should be allowed to handle a bottle of blood. The blood was grouped in the laboratory by an experienced technician who was supervising the work, and a check was carried out by the senior registrar or a registrar of experience. The bottle then passed, with the name of the patient and of the donor, to a central refrigerator, from which it was taken by a member of the medical staff. By this means the number of persons handling the blood had been reduced to the minimum—the one who checked and the one who gave.

Dangers of Domestic Refrigerators

In some general discussion Dr. T. V. COOPER pointed out the danger of using the ordinary domestic refrigerator for storing blood. In fact, unless there was a recording thermometer, any type of refrigerator might let one down very badly. Dr. J. C. BRUNDRET said that in Liverpool he had a fairly complex unit. He thought that to establish a rigid national procedure would be far more trouble than it was worth. Generally speaking, a separate technician was responsible for the checking system. Not only was the blood carefully labelled but a register went with it and signatures were required. He thought that the handling of blood could not be restricted entirely to medical people, but it should not go beyond the qualified nursing staff.

Dr. R. A. SLADDEN complained of the unduly large amount of blood which was used in many cases. Often, where two or three bottles would be adequate, eight or ten were demanded. When they were asked in an emergency to match up a bottle of blood, this could be done reasonably quickly, but if six or eight bottles were required it obviously made a great difference. Many surgeons had built up a system of using very large amounts of blood, and, as most patients responded well to treatment, the success had been credited to the massive transfusion, but no control series had been taken. He also referred to the difficulty of attending to blood transfusion demands in small hospitals in the middle of the night. There were not at present enough really skilled technicians to cover the needs of all the hospitals.

Dr. S. C. DYKE said that there was one aspect of this matter which ought to have a wider publicity. His own principle was to make it as difficult for anybody to get blood out of his bank as out of a stone, unless they had very good reason indeed. The occasions when large quantities of blood were required were relatively rare, and that was an aspect of the matter which he would like to impress upon clinicians. The ultimate giving of the blood was the clinician's responsibility, and he thought the clinician should have much more cognizance of the dangers of giving too much blood, one of which was liver damage.

A Standard Procedure

The CHAIRMAN said that there was one recurring theme in this discussion—namely, the desirability or otherwise of having a standard method of doing the whole of the cross-matching and so forth. He felt that this was a rather important matter, and, while it would never, he imagined, be true that any standard method could be followed in every case, he thought it would be helpful to have resort to such a method when necessary. A procedure might be laid down which was considered to be the ideal procedure, and any deviation from it should be a matter for very careful consideration.

Dr. HEGGIE said that in the North-eastern Metropolitan Region they had had in use for the last eighteen months a memorandum of instructions on the lines which the Chairman had suggested. They had a scheme which they had regarded as ideal, and also they had suggestions for agreed limitations and minimal degrees. He thought that if this were carefully followed they would have a perfectly valid defence in any court proceedings which might follow in a particular case. One little-known complication in blood transfusion was caused by organisms which might be present in bottles of blood but gave no sign of their presence by change of colour or by haemolysis. He knew of three fatal cases as a result of these organisms, the patients dying from massive protein shock.

Dr. C. R. LANE, Dr. J. G. A. MCSORLEY, and Dr. C. J. C. BRITTON also contributed briefly to the discussion.

Journal Article

Dr. J. M. ALSTON referred to the article under the heading "Blood Transfusion Accidents," published in the *British Medical Journal* of August 15 (p. 390). The article was published under the name of the Consulting Pathologists Group Committee, and he wanted to know how far the Group was committed by what was stated in the article.

Dr. E. E. CLAXTON (Assistant Secretary) explained that the Group was in no way responsible. The article was written by Dr. Heggie at the request of the Group Committee, but it did not mean that the Committee was responsible for what was stated in the article.

Dr. ALSTON said that he regarded the article as a very good one. He was not expressing any criticism of the article, but he did not wish it to be taken as committing the Group.

Dr. DISCOMBE, in replying on the discussion, agreed that the domestic refrigerator was a source of great trouble. One point had not been mentioned. In cold weather the refrigerator went down well below freezing-point, and he had had on occasion to put an electric radiator in his refrigerator. With regard to hepatitis, there were so many subclinical cases that he doubted whether any proper assessment could be made on how far this condition was due to previous blood transfusion. He did not think they had many extravagant demands for blood.

ADVANCES IN PATHOLOGY

The 51st General Meeting of the Association of Clinical Pathologists was held in London from October 1 to 3. Twenty-six papers were read and there were 11 demonstrations. In this report prominence has been given only to those contributions likely to interest the general reader. Not unnaturally at a meeting of this kind, most of the papers were concerned with specialist and technical problems. On the second day Professor Dorothy S. Russell delivered her presidential address on "Further Advances in Pathology."

A Relapsing Diarrhoea

Dr. A. G. SHERA (Eastbourne) described what he claimed to be a hitherto unrecognized type of relapsing diarrhoea, characterized by an explosive early-morning diarrhoea with offensive yellow stools which are often streaked with blood. It was caused by Vincent's organisms from chronic infections in the mouth and tonsils which passed the acid barrier in the stomach and settled in the rectum or sigmoid, forming characteristic "strawberry lesions." The way was prepared for the inception of these lesions by an associated vitamin-C deficiency. Diagnosis was by examination of stained faecal films or films made from swabs taken from the strawberry lesions, and by a vitamin-C saturation test. Treatment was specific, with acetarsol and ascorbic acid orally.

Colonel H. R. GILMORE (U.S.A.M.C.) described the epidemiology and clinical and pathological features of epidemic haemorrhagic fever ("Korean disease"). There was no evidence of spread from person to person or through water and food. Russian and Japanese work suggested that the causative organism is a virus or rickettsia carried by a mite, *Laelaps jettmari*, which infests the common field-mouse. In contrast, American workers preferred to incriminate a trombiculid mite, since *L. jettmari* is most prevalent in Korea, where the incidence of haemorrhagic fever is low. But they had not succeeded in passing the disease to animals or chick embryos, and the causative agent and its means of transmission were still undetermined. There were no specific laboratory tests for the disease, though albuminuria was a constant finding and 90% of cases showed increased capillary fragility. Post-mortem findings were haemorrhages into the kidneys, right auricle, anterior pituitary, and occasionally the suprarenals. Apart from these more dramatic findings, there were also submucosal petechial haemorrhages throughout the gastro-intestinal and respiratory tracts.

Dr. G. J CUNNINGHAM (London) described two cases of malignant hypertension in which aberrant renal vessels were found at necropsy. One of these aberrant vessels had become thrombosed, and the parts of the kidney supplied by it showed mild hypertensive arteriolosclerosis, while the rest of the organ, supplied by patent vessels, contained necrotic lesions of both arterioles and glomeruli.

Dr. A. A. G. LEWIS (London) described some studies he and Mr. L. P. LEQUESNE had made on the retention of salt and water in patients after surgical operations. The usual sequence of events in such patients might be divided into three main phases. During the first 24–48 hours after operation there was diminished water excretion, owing to release of pituitary antidiuretic hormone, probably as a result of emotional disturbances caused by the operation; this phase was accentuated by anaesthesia and the giving of morphine. Sodium retention occurred on the day of operation, and was due mainly to haemodynamic factors, although increased potassium excretion was associated with it. Thirdly, a second period of sodium retention usually began on the third day after operation, often separated from the earlier phase by a period of increased sodium excretion. The magnitude of the retention during this second period might be lessened by ensuring an adequate intake of potassium.

Limitations of the Test Meal

Dr. F. AVERY JONES (London) drew attention to the limitations of the fractional test meal in the diagnosis and treatment of gastric diseases, pointing out that the widely accepted normal range of acidity is based on findings in only 80% of Bennett and Ryle's series of healthy subjects. Achlorhydria was generally regarded as implying a gastric juice with a *p*H greater than 3.5, at which reaction, however, there was still about 0.3 mEq HCl per litre. There was little advantage to be gained from the routine use of test meals in cases of peptic ulcer, but they might help in the diagnosis of difficult cases with symptoms referable to the upper abdomen, in which radiological reports had been negative.

Dr. P. W. DARBY (London) described the results of a battery of liver function tests in 50 patients with rheumatoid arthritis and 45 hospital patients with non-hepatic disease; 23% of the rheumatoid and only 4.5% of the control group gave abnormal results to the bromsulphalein test, and 31% of the rheumatoid compared with 5% of the controls had an abnormal urinary coproporphyrin excretion. These differences were statistically significant, and suggested that there is a higher incidence of liver damage in patients with rheumatoid arthritis than in those with other forms of chronic ill-health. Drs. J. HARKNESS and C. DONOVAN (Portsmouth) confirmed previous reports that there is an increase in total ascorbic acid of the plasma with a greater proportion of reduced ascorbic acid during four hours after an injection of A.C.T.H. Injections of cortisone had no effect on the level of total ascorbic acid but did increase the proportion of reduced ascorbic acid. These changes could be used as a test of pituitary function, since by using an unvarying stimulus to the pituitary the ascorbic acid test indicated differences in individual responses. For example, the response in some psychotics was lower than in normal controls. Changes similar to those produced by A.C.T.H. were caused by electric convulsion therapy, emotional stress, and the administration of salicylates and phenylbutazone.

NEW ACADEMIC YEAR

FACULTY OF MEDICINE, SHEFFIELD

Professor L. J. Witts, Nuffield Professor of Clinical Medicine at Oxford, delivered the opening sessional address to the Faculty of Medicine, Sheffield University, on October 8. He took as his subject "The Opportunities of Medicine."

Professor L. J. WITTS said that he approached the task of speaking at an inaugural meeting in the spirit of Lincoln's Gettysburg address, and, rather than exhort the student, he wished to express the responsibility of the teacher, to whom so much precious human material was entrusted. There was no lack of enthusiasm for teaching on the part of the staffs of our hospitals and medical schools; the question rather was what they were to teach, and this was intimately linked with the nature and purpose of the medical profession.

The medical curriculum had not changed except by accretion since the early 1890's, when Osler and his colleagues at the Johns Hopkins Hospital started the combination of a university education in anatomy, physiology, and other sciences with an apprentice type of teaching in general

medicine and surgery. Those who asked for more training in general practice, dermatology, or psychiatry did not usually wish to disturb this general framework. More radical reformers, however, would substitute statistics, semantics, logic, psychology, and anthropology for some of the present basic sciences, and an even more revolutionary change was to reorganize the curriculum entirely, so that the basic sciences, pathology, and clinical medicine were taught simultaneously instead of in succession. In fairness it should be said that, with all its faults, the present curriculum does work. It was no good planning curricula which could be run only by completely dedicated teachers. For most teachers in medical schools, practice, research, and administration were the major preoccupations. This was right if we believed in the great didactic of Comenius, that our objective should be to seek and find a method of instruction by which teachers may teach less and learners may learn more.

The Status of Doctors

Despite the delightful pictures of Luke Fildes and Robert Louis Stevenson and the querulous letters in the medical press, there was no doubt that the medical practitioner was better off now than ever he was. The future maintenance of this status depended on our own efforts. Medicine must remain a learned profession, and it would be unwise for the universities to spend too much time on technological studies. Doctors are the most important group of biologists in the population, and in the recent war the contribution by doctors to greater efficiency in the fighting Forces and in industry was very great. This influence should be maintained in peacetime, for the great mass of the nation, including many of its leaders, was scientifically uneducated.

Medicine as a profession had three functions to perform —utilitarian, academic, and humanitarian. The humanitarian function required something corresponding to the minister's training in pastoral theology. It was doubtful if this was entirely a question of more hours devoted to the teaching of psychology and psychiatry. Patients and their relatives looked to the doctor for moral support in their hour of need, and most doctors, like their patients, still stood between two worlds, in a place where the orthodox creeds were no longer accepted but the need for a coherent philosophy remained. It was essential for the doctor to know something about religion, even if he did not believe it, and he should study the lives of great and good men, if only to carry out the Hippocratic injunction, "In purity and holiness shalt thou pass thy life." Much was learned from working with patients, for their heroism was often remarkable. Just as the doctor learned to transmit scientific knowledge, so it might be possible for him to transmit courage and serenity from his studies and his experience.

CHARING CROSS HOSPITAL MEDICAL SCHOOL

The inaugural session of Charing Cross Hospital Medical School took place at the neighbouring Royal College of Physicians on October 9. The chair was taken by Lord Inman and the address delivered by Admiral of the Fleet Sir Philip Vian.

Domiciliary Medicine for the Student

The Dean of the School, Dr. E. C. WARNER, reported a year of consolidation and progress. The level of success at examinations was high, and there were many individual distinctions. But the aim of the School, said Dr. Warner, was not so much to prepare the students for examination as to give them a grounding in the principles and practice of medicine so that they went out properly equipped for the work they had to do. To be a successful doctor something more was needed than learning: the human touch must be acquired. Arrangements had been made for students to attend domiciliary midwifery cases and also to be attached to general practitioners so as to see the family doctor's work at close quarters. One difficulty in a hospital like Charing Cross was to obtain experience in paediatrics owing to the smallness of the child population in the neighbourhood, but this had been met, at least in part, by association with a hospital in the East End of London. In view of building delays the intention of the hospital to move to a site outside London has so far not been realized.

Sir PHILIP VIAN gave an address in which he breezily related some experiences in the Royal Navy, both in peace and war. He said that his association with the medical profession had been chiefly with officers in the Royal Naval Medical Service, whom he had found uniformly kind, conscientious, and able men. For a short period during the war he had his own personal surgeon—a man who threw up his practice in Essex in his eagerness to serve in the Navy, and after several refusals by the authorities succeeded in persuading them to allow him to accompany Sir Philip Vian on his staff, and in that capacity took part in the Normandy landings. He also spoke of his pleasant association with Charing Cross, where his sister had until lately been on the nursing staff.

The vote of thanks was proposed by Professor W. J. Hamilton and seconded by Mr. H. G. Everard Williams.

BRITISH POSTGRADUATE MEDICAL FEDERATION

The inaugural lecture of the new session under the title "The Scientific Basis of Medicine," arranged by the British Postgraduate Medical Federation of the University of London, was delivered on October 8, in the Senate House, by Sir Henry Dale, O.M.

Need for a Broad View

The chair was taken by LORD WAVERLEY, who reminded his audience that the Federation, of which he was Chairman, embraced a number of scientific specialist institutes each providing higher instruction in a limited but clearly defined part of the general field of medicine and surgery. Since the tendency must inevitably be towards greater specialization it was becoming increasingly difficult for any student to keep abreast of the continually changing and developing basic knowledge in subjects outside his own specialty. It had therefore been decided four years ago to provide under the aegis of the Federation a series of lectures on the scientific basis of medicine. The lectures were intended for young research workers, primarily for specialists in training, and were designed to give them a knowledge of the scientific foundations of their subjects and to show something of the advance that was taking place.

Introducing Sir Henry Dale the Chairman said he was a doyen of medical science, to whom they would be happy to listen as often as he could spare the time to speak to them.

Sir HENRY DALE said he was rather diffident about giving this lecture, since it was almost identical with that which he had delivered three years ago on a similar occasion, and which had been published in full in the *British Medical Journal* (1950, **2**, p. 1186) under the title "Scientific Method in Medical Research." He had, however, been persuaded by Sir Francis Fraser, Director of the Federation, who had used the argument that the audience would be almost entirely different from the one of three years ago. Referring to the programme of lectures arranged for the new session, Sir Henry Dale said, "Knowledge is advancing at such a rate in so many parts of a widening field that the supply of special topics seem likely almost to outrun rather than to fall behind the number of lectures which a session like this could manage to accomplish." Little more than thirty years ago the position was that the only hormone treatment known was by thyroid gland, and no vitamins had been identified; less than twenty years ago there were no sulphonamide drugs and no antibiotics. It was almost impossible to think oneself back to the helpless position of medicine in the face of many infections at that time. On the other hand, he was inclined to think that the triumphs of therapeutics might have claimed so much attention that too little might have been said of other remarkable changes that had been taking place. "What impresses me more and more about

the present position is that at last the whole of medical research activity, whether in the ward or in the experimental laboratories, is moving forward on a common front."

Sir FRANCIS FRASER, in proposing a vote of thanks to the lecturer, gave it as his opinion that the new series of lectures could not have been better inaugurated than in the way they had heard that evening.

Nova et Vetera

THE ROYAL CHEST HOSPITAL

One by one the little lamps are being extinguished, and it will not be long now before the Royal Chest Hospital is snuffed out. Commonly regarded as the first chest hospital in the world, it owes its existence to the enterprise of one man.

Dr. Isaac Buxton (1775–1825), of New Broad Street, was struck by the fact that, although there were separate hospitals for many diseases, and although deaths from pulmonary complaints were extremely numerous, yet there was no establishment where the treatment of pulmonary disease might have a "fair and persevering trial." His own partiality was for a moderate summer temperature of 60–65° F. in the wards all the year round, combined with skilled nursing, and he founded the hospital principally for that purpose.

Its first full name was Infirmary for Asthma, Consumption and other Pulmonary Diseases. In the course of time there were several variations on this theme, and in 1848 Queen Victoria bestowed the Royal title. Later the "Heart and Great Vessels" was also incorporated in the name, and cardiovascular disease has had its place in the hospital work ever since. In 1919 all the activities were telescoped into the title of Royal Chest Hospital. Sir Astley Cooper was interested and helped to secure the Duke of Kent as President, and in 1814 the Duke opened the Infirmary at 36, Union (now Brushfield) Street, Bishopsgate. It was a twelve-roomed house on a site now occupied by a tavern at one corner of Spitalfields market.

In 1832–3 it went first to No. 13 and then to No. 10 Artillery Street (now Lane) for fifteen years, to premises previously used as a dispensary. The house was in such a condition of insanitary squalor that for three years Dr. Ramadge, physician in charge, refused to admit in-patients, but he could not persuade the committee to improve the drains or water supply. In 1847–8 there was another move to 4, Dean (now Sun) Street, Finsbury Square, for about a year. Then in 1849 the hospital finally settled in the City Road in a house belonging to St. Bartholomew's Hospital.

Shepherds at the Angel

At that time residential terraces were springing up in the City Road, and there were already three hospitals on its south side. To the north, behind the new hospital, there were green pastures, and shepherds still guided their flocks through the two turnpikes in the road and refreshed themselves at the Angel Inn. Islington Spa and the entertainment halls in the locality were popular, but not so fashionable as they had been.

In 1862 the house was pulled down and a building more suitable for hospital use was erected. In 1876 further building was done to provide for the increasing number of out-patients, and in 1880 St. Bartholomew's sold their freehold and further extensions were made, completing the frontal appearance familiar to us with the later addition of a nurses' home and a pathological department behind.

In 1912 the tuberculosis dispensary for the boroughs of Islington, Shoreditch, and Finsbury was housed in the hospital, an arrangement which earned special praise from William Osler. In 1919 the hospital amalgamated with the Great Northern Hospital, which paid its debts and acquired a title, leaving the medical staff to continue their work independently and unimpaired until the second world war, when the whole of the front portion was destroyed by a bomb in 1941.

Pampered by Royalty

For material support in the early days the infirmary had the interest of the City Corporation, the City companies, and the City magnates, and for patronage it had the Royal Family. It was positively pampered by Royalty. Three Royal dukes in succession were presidents; Queen Victoria subscribed liberally and became a patron at a low ebb in its fortunes; other members of the Royal Family visited, and Princess Louise laid a foundation stone in 1876. Three princes of the blood presided at different festival dinners for raising funds; these were usually held at the celebrated London Tavern, Bishopsgate, where the ladies were callously relegated to a watching gallery until one day a rebel in the shape of Dr. Horace Dobell flouted the angry conservatism of the stewards and a very jolly evening ensued, with a benefit of £1,000 for the hospital.

The Early Physicians

Some of the early physicians were interesting personalities. Dr. Buxton started life as a nonconformist minister, then graduated in medicine at Göttingen. He was physician at the London Hospital for fifteen years, but there seem to have been strained relations with the governors at the time of his resignation. He succeeded in persuading several members of his philanthropic family to help the infirmary in different ways.

Dr. F. H. Ramadge followed Dr. Buxton as sole physician in 1820, and he also was possessed of a pioneer spirit. He very soon instituted a regular course of postgraduate lectures at the infirmary and started assembling a pathological museum, both of which pursuits must have been unusual at a non-teaching hospital in those days. It has been said that Ramadge was one of the first advocates of artificial pneumothorax for consumption, but the truth is just the opposite. He invented an inhaling tube for keeping the lungs well inflated, and he published many papers on the curability of consumption on those lines. He may have exaggerated the value of his instrument, but his methodical necropsy work had rightly convinced him that consumption was not so invincible a disease as most of his colleagues believed. Dr. Ramadge had disapproved of the Artillery Street house from the first, and was on very bad terms with the committee in consequence. After an exchange of offensive letters from both sides he yielded to pressure and resigned in 1847, full of resentment.

Dr. Thomas Davies was elected additional physician in 1825, and he was perhaps the most successful of the early medical staff. Having worked under Laennec for two years, he was adept at auscultation, and he did a great deal to promote the rational use of the stethoscope among practitioners in the City between the years 1825 and 1839, when he died. He also designed the first thoracic exploring needle in order to spare patients from paracentesis for purely diagnostic purposes. His postgraduate lectures were so renowned that the London Hospital invited him to repeat them and made a place for him as physician. They make good reading even now.

Dr. B. G. Babington was an early member of the committee, though not on the medical staff. In spite of this close connexion with the man who invented the glottiscope in 1829, it was not until 1866 that the hospital bought a laryngoscope, as the finished instrument was eventually named.

Subsequent physicians and surgeons maintained the reputation of the hospital, which already in 1862 was noted to be drawing patients from the home counties, the north of England, and Scotland. Among the names (other than those still alive) we find A. Leared, Herbert Davies, W. Hale-White (the first pathologist), Jonathan Hutchinson, A. Pearce Gould, Bertrand Dawson, and Symes Thompson, to whom I am indebted for some of these notes.

Teaching and Experiment

The postgraduate teaching started by Dr. Ramadge in 1822 has continued, off and on, ever since. In 1883 a medical school was included in the plan of the new building,

and in 1889 there was a public row when the medical staff were accused of using the hospital more for teaching and experimental purposes than for relief of the sick poor. One of the incidents in dispute was Moore's wiring operation for an aneurysm by Mr. Pearce Gould; another was an emergency tracheotomy performed by a house-surgeon.

Mr. Austin, the secretary of that period, was very popular with the humbler citizens of the district, who were fond of their cosy little hospital round the corner, and with his encouragement they collected one-sixth of its income, mostly in pennies. It was probably on his initiative that the board was progressive enough to admit representative "working men" as governors on a collective subscription of £10 from a group.

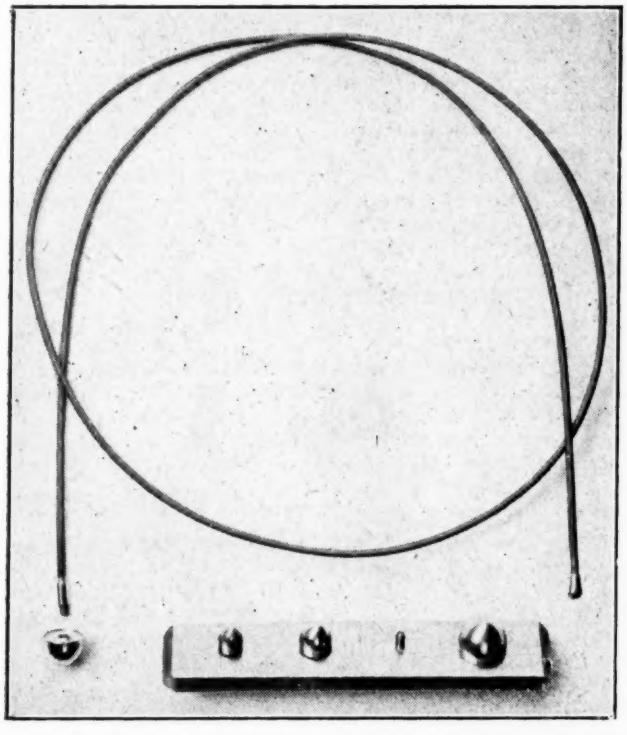
The Royal Chest Hospital has never had many beds. It started with 8 and ended with 80. There have been intervals when there were no in-patients at all, and in the last thirteen-year period after the wards were damaged and never repaired it has been possible to attend only to out-patients. The hospital has enjoyed the advantages of a compact, enthusiastic unit and suffered the disadvantages of smallness, whatever they may be. Next year nothing will be left except the scorched earth, a crowd of ill people, a few frustrated consultants, and some faithful members of the staff.

NORAH H. SCHUSTER.

Preparations and Appliances

A VEIN STRIPPER

Dr. ELIAS N. TSARDAKAS, Whittington Hospital, N.19, writes: In the modern and popular operation of vein stripping I have found the following modification of the Babcock instrument of great use. It has the following advantages: (1) The varying sizes of head allow for the wide range in the diameter of veins encountered; (2) the



great flexibility of the spiral spring stem—24 in. (60 cm.) long and silver-coated—facilitates the removal of veins, no matter in what direction they run; (3) the whole apparatus is very easily sterilized and stored, and will fit into an ordinary cigarette packet of 20; (4) it is manufactured very cheaply. The vein stripper has been made for me by Messrs. Allen and Hanburys Ltd., Wigmore Street, W.1.

Correspondence

Because of the present high cost of producing the Journal, and the great pressure on our space, correspondents are asked to keep their letters short.

Cancer and the Public

SIR,—Dr. J. F. Brailsford writes (*Journal*, October 3, p. 779): "One might think from the propaganda enthusiasts that there was a cure for cancer. . . . Whereas the truth is that we have no cure." He must know that for years it has been customary to talk about the results of treatment in terms of five-year "cures" or ten-year "cures," the word implying that the patient is apparently free of the disease over the period stated. It seems to me rather mischievous to infer, as Dr. Brailsford does, that the propagandists are misleading the public by telling them that cancer can be cured. What they do keep on telling the public is that treatment of cancer is improving and they should know about it. It is rather difficult always to avoid using the word "cure"; one is entitled to speak of the chances of being cured rather than in terms of "the percentage survival with no evidence of the disease." Few people are really interested in percentages.

Dr. Brailsford hardly serves his case by quoting Shakespeare, for the patients who had knowingly had cancer 7–10 years but failed to consult a doctor had indeed let cancer "feed on the pith of life." The poet saw the danger centuries ago, but we are still boggling at the idea that the public should be told some of the most pertinent facts about cancer.—I am, etc.,

London, W.C.1. SIDNEY RUSS.

SIR,—The counsel of despair implied in Dr. James F. Brailsford's letter (*Journal*, October 3, p. 779) cannot be allowed to pass unchallenged, and one wonders whether it really represents his considered opinion. He must know that the fear of cancer is already present in the minds of the public just as the fear of having an abnormal baby is in the thoughts of every pregnant woman. It is to allay such fears that education may be of service.

When the figures of the British Empire Cancer Campaign's inquiry are analysed it will be seen that three-quarters of the doctors consulted did not even reply. To say, therefore, that the majority of general practitioners are opposed to lay education when only about 1 in 8 gave such an opinion is surely a false use of statistics.

Those who see many cases of cancer, however, would welcome any method of encouraging patients to seek treatment early, and if lay education can do this we would welcome it, for without holding out hopes of absolute cure quite a proportion of patients are spared considerable suffering for many years. No exaggerated claim has, in fact, ever been made for lay education; it is evident, however, that the lay mind must be educated to realize that what happened in the treatment of cancer 100 years ago should not be accepted for to-day. Or is Dr. Brailsford prepared to discredit the statistics of so many reliable centres all over the world? He surely would not advocate that patients with cancer should retire to some secluded spot and allow it unmolested "to feed on the pith of life." The reference to the curability of syphilis is somewhat inapposite, for surely neurosyphilis may be completely incurable even with the most violent treatment. Quite obviously the search for a cure for both syphilis and cancer must go on, but until then we must use every available method of treatment and suitable propaganda to combat them. Dr. Brailsford would surely subscribe to Montaigne's dictum, To cure sometimes, to relieve often, and to comfort always.—I am, etc.,

Pembury, Kent. E. D. GRASBY.

SIR,—To suggest, as Dr. J. F. Brailsford does (*Journal*, October 3, p. 779), that the removal of a carcinoma, without recurrence and with a patient leading a normal active life,

is no cure seems to me to be a terminological hair-splitting quibble. Two years ago I had a partial colectomy for a carcinoma of the colon. I live a perfectly normal life, and practise as a busy consultant surgeon. I am free of all symptoms. To suggest that I might as well not have consulted a surgical colleague because no cure can be held to occur merely by the removal of the affected area is, to me, just nonsense. I hope that others may benefit as I have, and nothing should be said or written to discourage them from seeking advice as early as possible. For obvious reasons I prefer to sign myself

A Grateful Patient.

Television in Health Education

Sir,—The report of the General Practice Review Committee (*Supplement*, September 26, p. 103) includes some strong adverse criticism on the use of television in health education. The fact is that television is an extremely potent but delicate weapon for health education, and must be handled with great skill or its effect on the public will be deplorable.

If, as the report stresses, the use of this medium has not always been happy, it seems ungrateful not to mention at the same time how valuable a contribution television can make when expertly used, as in the three brilliant documentaries written by Dr. Cormac Swan—"Dangerous Drugs," "Family Doctor," and "Under Her Skilled Hand"—each of which perfectly fused instruction and entertainment and did the job they were designed to do.

I should be sorry to see a medium potentially so powerful in this field condemned and cast aside through a failure to learn from these documentary models.—I am, etc.,

London, S.W.1. Guy Wigley.

Orthopaedic Care in Poliomyelitis

Sir,—Like Sir Reginald Watson-Jones (*Journal*, October 10, p. 828), I am glad that physicians, laryngologists, and anaesthetists are making so determined an effort to save the lives of patients severely stricken with poliomyelitis. At the same time I share his disquiet at the apparent tendency to equate the early care of the patient in danger of his life with the whole treatment of the disease. I, too, have seen the Welsh Regional Hospital Board's circular, and it is a disturbing document. Undoubtedly we must be prepared for a catastrophe such as has stricken Copenhagen—and it happens that part of the work on the design of the British cuirass respirator is being undertaken here by my colleagues Dr. Kinnier-Wilson and Dr. J. C. Scales. But I am beginning to wonder whether, in anticipating an epidemic of a kind that has occurred only twice in the history of poliomyelitis, there is a danger of neglecting to strengthen our resources for dealing with the disease as we find it in Great Britain and as it has occurred this year. In Coventry, for example, there has been only one death and only four patients requiring respirators. Even in Copenhagen the ordinary manifestations of poliomyelitis, paralysis of the limbs and trunk, and the ever-present tendency to contracture and deformity, were and still are important.

May I try to illustrate what I mean by reference to but one aspect of the early treatment of poliomyelitis—namely, the prevention of deformity? This word does not appear in the Welsh Board's document, yet the title is "Poliomyelitis" without qualification. Nevertheless, the prevention of contractures and deformity is of immense interest to those primarily concerned with their prevention and treatment—namely, orthopaedic surgeons. It was W. J. Little, a physician on the staff of the London Hospital, who, in 1838, founded the first orthopaedic hospital in London. He was well informed about the mechanisms of deformity, and wrote a remarkable book on those affecting the foot. There was a good reason for this; he himself had a deformity of the foot that was almost certainly caused by poliomyelitis. But I have not yet met a twentieth-century W. J. Little. Those interested mainly in the acute disease see before them a patient who has paralysis and who is perhaps gasping for breath and unable to swallow; at this stage his joints are not stiff and his body is not misshapen. They are but dimly aware of what lies ahead; they have not seen the grotesque deformities that can occur; they do not appreciate the significance of, say, a little stiffness in the joints of the fingers.

Twenty years ago I saw many operations performed on bones, joints, and tendons for correction of paralytic deformity. Most of these operations are now obsolete. It was increasingly recognized that deformity can easily be prevented if what for the sake of brevity may be called orthopaedic treatment is started before contractures can develop—that is to say, in the first weeks of the disease. Furthermore, if late deformity was to be avoided, supervision of the patient was necessary over a period of years. Gradually a vast apparatus for dealing with crippling conditions, of which poliomyelitis is now one of the most important, was built up: the orthopaedic establishment (there are no fewer than 43 special hospitals) with its physiotherapy department and pool, plaster room and splint shop, hospital school, rehabilitation scheme, and follow-up clinics. Are we to understand from our brethren in Wales that all this is now of no consequence, or perhaps something taken for granted like the supply of electricity or water? If the orthopaedic surgeon and what he represents are excluded—as was done in Cardiff—a most vital element is missing.

In the report of the meeting mention was made of additional facilities, and no doubt they are needed. But, whatever form an epidemic may take, the place where additional facilities are most urgently required is the orthopaedic hospital. At the present time most orthopaedic surgeons are handicapped by long waiting-lists and by inadequate assistance. Yet theirs is the ultimate responsibility. The patient will come to the orthopaedic department or hospital at last, however many sorts of people may have dealt with him on his way there. I once visited a capital city of northern Europe where the role of the orthopaedic surgeon was to take charge of the victims of poliomyelitis at a time dictated by the physician who treated them in the early stages. I found the surgeon I met there was far busier with his cases of poliomyelitis than we are accustomed to be. He was expert in the sort of corrective procedures that most of us here had hoped never to practise again. Are we in Great Britain to go the same way? Already there are a few signs of it. They are there to be seen in the wards of our country hospital at Stanmore. We estimate that inefficient treatment in the early stages lengthens the patient's stay in hospital by one-third to one-half.

This interest in bulbar paralysis is welcome, yet although there is the potential threat in every case, the plain fact is that most patients with poliomyelitis escape it. It is becoming difficult now to hear the still small voice of common sense above the whirring of pumps and the whistling of tracheotomy tubes. Would it not be better to make a concerted effort to provide better treatment for every aspect of poliomyelitis, each member of the team contributing his own special skill for the benefit of those who have fallen victims to this dreadful disease?—I am, etc.,

London, W.1. H. J. Seddon.

Sir,—It is regretted that the British Orthopaedic Association should have gained the impression, as suggested in Sir Reginald Watson-Jones's letter (*Journal*, October 10, p. 828), that orthopaedic surgeons are in any way excluded from their rightful place in the treatment of poliomyelitis in Wales. It is only fair to point out that the special committee of consultants which met some time ago were dealing essentially with one aspect of poliomyelitis, that of the respiratory complications.

The recommendations in the memorandum to which Sir Reginald refers are additional to the already existing arrangements, which are described and which make special mention of the necessity for orthopaedic supervision.—I am, etc.,

A. Trevor Jones,
Senior Administrative Medical Officer,
Welsh Regional Hospital Board.

Cardiff.

Succinylcholine Chloride for Casualty Anaesthesia

Sir,—We should like to suggest the following technique as a simple and effective method of obtaining adequate relaxation during manipulations in the casualty department.

A syringe, containing 100 mg. of succinylcholine chloride ("scoline") and fitted with a small-bore needle, is introduced into a vein on the dorsum of the hand and strapped in position. The hand is then placed across the patient's chest, where it can easily be reached by the anaesthetist while he is still holding the face mask in position. Light anaesthesia is obtained with nitrous oxide and oxygen from a Boyle's apparatus. As soon as consciousness is lost, 25 mg. (for an average healthy adult) of succinylcholine chloride is injected, and approximately 15 seconds later the surgeon commences reduction. This dosage gives excellent relaxation without causing complete apnoea. By the time the plaster has been applied, completely normal respiration is re-established and the anaesthetic is discontinued. The patient is now able to walk out of the theatre and may safely go home. Should momentary apnoea occur, this is readily dealt with by gentle compression of the rebreathing bag. If a check *x* ray is required, the patient is maintained on gas and oxygen while films are taken and developed. Should further correction prove necessary, the dose of succinylcholine chloride may be repeated.

We have used this method with success in small children, reducing the dosage of succinylcholine chloride according to the patient's size.

This method is particularly valuable in a busy casualty department where the shortage of nurses and recovery rooms renders it necessary to send the patient home as soon as possible.—We are, etc.,

Terence Vaz.
J. M. Bishop.

Slough.

Bornholm Disease and Poliomyelitis

Sir,—Dr. Gilbert Dalldorf (*Journal*, August 1, p. 287) asks whether poliomyelitis and Bornholm disease ever occur simultaneously. As Bornholm disease is not notifiable, evidence on this point is probably scanty, so the following may be of interest.

Since the beginning of 1952 a group of 10 general practitioners, strategically selected to cover the city of Auckland, have been reporting to me every week details of non-notifiable infectious diseases seen in their practices. Their figures represent about one-fifteenth of the cases attended by doctors in the population of 302,560 within 10 miles of the centre of the city. The table below shows the actual numbers of cases of Bornholm disease seen by this group, as compared with the number of poliomyelitis cases occurring in the same area.

Month	B'holm	Polio.	Month	B'holm	Polio.	Month	B'holm	Polio.
1952			1952			1953		
Jan. . .	9	0	Aug. . .	3	41	Feb. . .	8	9
Feb. . .	22	4	Sept. . .	1	49	Mar. . .	0	7
Mar.	18	5	Oct. . .	2	69	Apr. . .	2	4
Apr. . .	4	5	Nov. . .	0	45	May . .	1	7
May . .	4	0	Dec. . .	2	38	June . .	6	0
June . .	1	2	1953			July . .	5	0
July . .	2	20	Jan. . .	0	17	Aug. . .	2	2

It will be noted that Bornholm disease waned as the poliomyelitis epidemic developed, and has shown a tendency to recrudesce as the epidemic subsided. The last case of poliomyelitis previous to February, 1952, occurred in April, 1951. The poliomyelitis incidence in the city on the present occasion reached 107 per 100,000.—I am, etc.,

Auckland, New Zealand.

A. W. S. Thompson,
Medical Officer of Health.

Poliomyelitis Vaccine

Sir,—The international committee on poliomyelitis, which recently met in Rome, has focused attention on the need for an effective vaccine against this disease. If it were ever to be shown that poliomyelitis and any other disease consistently occurred in different areas, or were in any way mutually exclusive, such a discovery would intensify the hope that we might eventually find a virus which stands in relationship to poliomyelitis as does vaccinia to variola. This would indeed be one of Nature's kindest gifts.

Every summer and autumn, nowadays, we are called to see a higher proportion of the fevered in our practices than used to be the case. Behind each such call is the fearful hope that "it is nothing really serious." Dr. A. P. Grimbly (*Journal*, September 12, p. 628) has recently stressed the importance of realizing that many of these cases may in fact be non-paralytic poliomyelitis, even in the absence of an epidemic of paralytic cases. Dr. Gilbert Dalldorf (*Journal*, August 1, p. 287) has asked for reports of any evidence that Bornholm disease and poliomyelitis do not commonly occur in the same district at the same time. Of equal importance would be reports from areas, consistently free from poliomyelitis, about the nature and epidemiology of any outbreaks of pyrexia of unknown origin and transient duration which occur about the same season as poliomyelitis. For from such information a clue might come to show that non-paralytic poliomyelitis and paralytic poliomyelitis, though antigenetically similar, were sometimes separable. This could happen if a viscerotropic variant of poliomyelitis virus exists and was ever distributed in epidemics under natural conditions.

Its existence could only be recognized in an area where it had succeeded, by prior immunization, in "blocking" the spread of the normal neurotropic virus. I believe that if an intensive survey could be carried out of the unknown pyrexias of summer and autumn, particularly in relation to those villages or groups of villages and small towns where paralytic poliomyelitis has not appeared since the war, we might find a natural viscerotropic virus which could better serve our purpose as a starting point for a vaccine than would normal neurotropic virus derived from paralytic cases.

Dr. R. Wigglesworth (*Journal*, August 29, p. 507) calls for an intensification of research into the ecology of a number of virus diseases, including poliomyelitis. In collecting information of this sort the College of General Practitioners might play a most valuable part.—I am, etc.,

Peaslake, Surrey.

G. I. Watson.

Cortisone and A.C.T.H.

Sir,—A widely read report on cortisone and A.C.T.H., based on two articles published in the "Annual Advances in Treatment" number of your contemporary *The Practitioner*,[1 2] appeared recently in *The Times* (October 2). We feel that these articles express inadequately the true status of these drugs in the modern treatment of rheumatoid arthritis. The authors state correctly that cortisone can have a profound and gratifying effect in suppressing the symptoms of rheumatoid arthritis. They go on to say, however, that after early hopes and optimism disappointment inevitably follows. After more than three years of experience, during which time we have returned many previously incapacitated patients to an active life on relatively small doses, we feel that this second statement is no longer true.

Both articles state that prolonged treatment may bring with it a host of difficult and even dangerous side-effects, mentioning addiction, fluid retention, cardiac embarrassment, and mental change—as well as others which we find more hypothetical than real, such as spontaneous fracture. The art of medicine would seem to us to be concerned with obtaining the effects desired while avoiding unwanted sequelae, and prolonged and unskilful treatment with many other drugs might also justify these statements. We agree that cortisone is a more difficult drug to use properly than was at first hoped, and this has discouraged many on both sides of the Atlantic who like fool-proof weapons. We have found it possible to achieve success, however, in a majority of cases if enough time and trouble are taken.

The authors also decry treatment with these substances on the grounds that quiescent infections, such as tuberculosis, may be masked, hypertension made worse, or a rebound of the disease occur on stopping treatment. All these possibilities should be well known to anyone using cortisone or A.C.T.H. Such infec-

tions must be excluded before cortisone or A.C.T.H. treatment is considered, and, if found, are absolute contraindications to their use. Treatment never should be stopped abruptly, but tailed off very gradually.

Readers are also advised against using these substances in rheumatoid arthritis, as "there is some evidence that in the long run the ultimate state of patients controlled by cortisone is no better than those controlled by adequate doses of aspirin." This amounts to saying that both drugs suppress but do not cure the disease. In those cases where such suppression can be adequately produced by aspirin we would agree that this should always be our drug of choice. It is only in cases where simpler drugs fail that cortisone is indicated. The authors conclude that "before using cortisone it is necessary to balance carefully the beneficial effects . . . against the risks which its use involves." Such advice is surely applicable when we consider the use of any powerful therapeutic substance. They also both suggest that "the early beneficial effects may need progressively larger doses for its maintenance." This statement is entirely contrary to our observed experience.

We believe that the modern status of cortisone and A.C.T.H. in the treatment of rheumatoid arthritis would be more accurately stated as follows. These drugs constitute the only short-term therapeutic weapons we possess with which we can, in many cases, completely suppress the signs and symptoms of this disease. They are not applicable to all cases—indeed, candidates for such treatment must be carefully selected, as there are many known contraindications. These substances are by no means foolproof, and considerable experience in their use is required if good results are to be maintained. Patients on such treatment must be constantly reviewed, as their therapeutic needs and responses may vary rapidly. Such treatment should be used together with the other basic forms of treatment designed to prevent deformity, etc., and never as the sole therapeutic method.—We are, etc.,

W. S. C. Copeman.
O. Savage.

London, W.6.

References

1 Dunlop, D. M. (1953). *Practitioner*, 171, 343.
2 Tegner, W. (1953). Ibid., 171, 424.

Danger of Penicillin Injection

Sir,—The reports of severe anaphylaxis following injections of penicillin are extremely interesting, and this condition is undoubtedly becoming more common (*Journal*, October 3, p. 770 and 777). I have seen one severe reaction in a nurse, following an injection of 200,000 units procaine penicillin.

Sulphonamides could be used in very many cases where penicillin and other antibiotics are now given. There is no doubt whatever that if sulphonamides had been discovered after the antibiotics they would have been regarded as a tremendous advance. Surely we should use sulphonamides much more frequently than we do, for they cause less inconvenience to the patient and the nursing staff, and are likely to give rise to less severe toxic reactions.—I am, etc.,

London, N.W.10. Horace Joules.

Spectacles

Sir,—The recent letters by Drs. R. U. Gillan and Christopher Heath (*Journal*, September 5, p. 568) concerning the probable or possible connexion between migraine and errors of refraction remind me of a wise saying by my late revered chief, Dr. Samuel Gee, which may be not without interest to your readers. Dr. Gee was a lifelong sufferer from severe and quite erratic attacks of migraine. He frequently consulted ophthalmological colleagues and they invariably prescribed spectacles. I have often heard him say that while he wore no glasses he never knew at what moment he might be prostrated by an attack. "When, however," he would add in that curious hesitant method of speech which it is quite impossible to reproduce in print, "I wore the spectacles which they ordered for me I was quite certain that I should be laid up by an attack within half an hour." It was Dr. Gee's invariable custom during a great many years to commence his rounds at St. Bartholomew's at 1.30 p.m. every Tuesday, Thursday, and Saturday. It is a curious and perhaps not uninteresting fact that during this long period he was never once prevented by an attack of migraine from carrying out this invariable routine.—I am, etc.,

Esher. C. F. Hadfield.

Myxoedema Coma

Sir,—Dr. V. K. Summers in his interesting article (*Journal*, August 15, p. 366) states: "Once coma develops (in myxoedema) it seems to be irreversible." The following two cases show that this is not always so and that recovery may be spontaneous.

A 63-year-old housewife was admitted in December, 1951, having lost consciousness a few hours before. She was grossly myxoedematous and was in coma, though responsive to stimuli. There was nothing else to account for the coma. The patient spontaneously recovered full consciousness within 24 hours of admission without specific treatment. A typical history of myxoedema was then obtained extending over two years. For six months she had been increasingly lethargic, drowsy, and sensitive to cold. The radioiodine excretion pattern was hypothyroid in type and did not alter materially after a course of thyroid-stimulating hormone. Her ketosteroid excretion in 24 hours was 5.1 mg., kepler factor A was 143. The patient remained fully conscious while these investigations were being done. She steadily improved with *l*-thyroxine therapy and was euthyroid three months after starting treatment.

The second case was a woman of 69 who was seen at her home in 1949. She was immensely fat, obviously in an advanced stage of myxoedema, and stuporose. Her trunk felt almost as cold as the surrounding January air. She had been vomiting for a few days and the blood pressure was 90/70–85/60. In spite of immediate admission to the hospital and prompt oral treatment with thyroid extract, stupor deepened to coma and she died five days later without recovering consciousness. Necropsy revealed almost complete atrophy of the thyroid. The pituitary was normal. there were large medullary haemorrhages in both suprarenals, but apart from recent compression the cortices were normal. The patient's history was obtained in detail from her daughter with whom she lived. She had clearly suffered from myxoedema for some years. The point of interest was that two years before her terminal illness the patient had been in coma at home for several days and had made a spontaneous recovery. It seems reasonable to suppose that on that occasion the coma was due to the myxoedema. In her last stages the vomiting and hypotension were probably due to the unusual occurrence of suprarenal haemorrhages which must have hastened the end.—We are, etc.,

Cardiff. L. Brotmacher.
Isleworth. N. F. Coghill.

Appendix in the Hernial Sac

Sir,—Mr. T. E. Winckworth's letter (*Journal*, September 5, p. 567) records a case of acute perforative appendicitis in a hernial sac in a man of 62, and inquires as to the frequency of this condition. It may be of interest, therefore, to report its occurrence at the earlier extreme of life. In March, 1951, there was admitted to hospital a baby of 7 weeks, with a tender irreducible swelling occupying the right inguinal canal. An occasional lump had been previously noted in this place by the nurse. The child had vomited once about 48 hours before, and after a normal motion had been constipated for this period. The temperature was 102° F. (38.9° C.), pulse 120, abdomen soft, both testicles normal in size and situation.

At operation on a diagnosis of strangulated inguinal hernia, a foetal type appendix perforated at its middle was found in a sac full of pus, the neck of the sac being shut off by the caecum. The appendix was removed, the stump being easily invaginated. The neck of the sac was closed,

and the canal was drained. The child was discharged healed on the tenth day, and has remained well since, having passed through an attack of pertussis without any sign of reherniation.—I am, etc.,

Minehead. H. B. Walker.

Sir,—I was very interested in the case described by Dr. T. E. Winckworth (*Journal*, September 5, p. 567), because I have on two occasions seen a similar condition associated with femoral hernia.

Two years ago, while I was working with the late Mr. W. E. Tanner, he operated on a woman for a femoral hernia which had recently become tender. At operation the sac was found to contain the tip of the appendix, which was considerably swollen and congested. There were early signs of inflammation, but the congestion was considered to be secondary to the impaction of the appendix in the neck of the sac. Curiously, exactly one week later, I personally operated on an identical case in a woman. I have not, however, heard of any similar cases.

With regard to the occurrence of the normal appendix in a hernial sac, I recently found one in the sac of an inguinal hernia in a small child.—I am, etc.,

Birmingham. D. G. Maurice.

Possible Sequel of Treating Infants in the Erect Posture

Sir,—I was interested to read Professor John Craig's reference (*Journal*, July 11, p. 98) to flattening of the head noticed in infants nursed in an upright posture. At this hospital a similar condition has been present in three cases of cardiochalasia or hiatus hernia nursed in a "chair." In one child the flattening was marked during the period of treatment, but at the age of 2½ years the shape and measurements of his head seem to have reverted to normal. He has not required any form of therapy since he was a year old.—I am, etc.,

Pontypridd. W. Davies.

Pain of Transurethral Manipulations

Sir,—As a urologist, I heartily agree with your two correspondents, Dr. E. C. Atkinson (*Journal*, August 22, p. 440) and Mr. H. Calvert Dales (*Journal*, September 5, p. 567). May I put in a plea for the use of smaller instruments as well as lignocaine ("xylocaine") and/or general anaesthesia?

In my clinic I use a 16 French viewing cystoscope, which I had specially made. This is only three-quarters the size of the 21 French cystoscope generally used. For double catheterizing I use either an 18 or 21 French American Brown Bueger instead of the 23 French double catheterizing cystoscope in general use, some of which in practice tend to be even larger than the listed scale. For anyone who is accustomed to endoscopy these smaller instruments give an adequate view, and I am sure contribute considerably to the comfort of the patient, both during the procedure and afterwards.—I am, etc.,

Luton. Henry Clarke.

Sir,—I am glad that Dr. E. C. Atkinson (*Journal*, August 22, p. 440) has raised the question of anaesthesia for transurethral instrumentation. It is a matter of astonishment to me that even now some surgeons pass instruments per urethram without any anaesthetic at all. I think that there is a tendency to decry local surface analgesics as useless because they are never given a chance. I have seen surgeons use a local analgesic, and immediately start to operate. Mr. Lang Stevenson waits for ten minutes, and Mr. Winsbury-White for five minutes (*Journal*, September 19, p. 674). I am sure it would surprise many readers to know how long five minutes' waiting means. If checked by a watch it is very much longer than one thinks.

For many years I have followed the advice of my colleague, the late Mr. Douglas Crow, who was in charge of the throat department at the Royal Sussex County Hospital. He used to teach that after anaesthetizing a patient with a local analgesic one should treat another patient, and after that return to the patient one has anaesthetized—by then the analgesic will have taken effect. I am sure that, if adequate time were allowed, local analgesics would be more effective, and Mr. Crow's simple method ensures this.

No surface analgesic will anaesthetize the deep structures, and gentleness is still necessary. I have had patients attending my out-patient clinic for years for dilatation of a stricture under a local analgesic, who have occasionally had to submit to this treatment without an analgesic when I have been away, and they tell me the local analgesic makes a very great difference. I follow the technique of the late Canny Ryall, but, having had a few alarms with cocaine, for many years now I have used 1/1,000 cinchocaine ("nupercaine") with good effect and no untoward results.—I am, etc.,

Hove. H. J. McCurrich.

Neonatal Asphyxia

Sir,—Your annotation (*Journal*, August 8, p. 328) and the correspondence on this subject have prompted me to quote an article describing a resuscitation trolley that was photographed and produced in the *Journal of Obstetrics and Gynaecology of the British Empire* (1949, 56, 1044).

This referred to a resuscitation trolley the purpose of which was to facilitate the passage of an endotracheal tube so that a newborn baby might be insufflated with oxygen and respirations established. Since this time in the last 3½ years this trolley has been present at every caesarean section (215) and forceps delivery, and a large percentage of the babies have required this method of resuscitation consisting purely of the rhythmic insufflation of the lungs through an endotracheal tube with oxygen controlled at the correct pressure with a water manometer. No other method of resuscitation has been used, and no drugs of any kind have been administered to a baby to stimulate respiration.

With the use of a resuscitation trolley as described it soon becomes practicable for an anaesthetist or obstetrician to pass an endotracheal tube in any sized baby, and with the use of a water manometer no injury to the baby will occur by too great a pressure of oxygen being applied to the baby's lungs.—I am, etc.,

Workington, Cumberland. James B. Joyce.

Sir,—I am grateful to Mr. W. G. Mills (*Journal*, September 19, p. 675) for writing that my "arguments are sound" and that my "practice can be shown to give dramatic results by anyone who takes the trouble to try," but I cannot claim the special skill with which he credits me in giving intracardiac injections. They are, in fact, simplicity itself in infants.

All that is required is a hypodermic syringe with a 1-in. (2.5-cm.) needle of fairly fine bore—a No. 15 or 16 is suitable. This is inserted about 3–4 cm. internal to and superior to the position of the normal apex beat, and slight suction is applied from time to time as it is inserted the last half of its length. Immediately blood appears in the syringe the desired dose of "cardiazol" ephedrine is injected. If by any chance the ventricular cavity is not struck the first time it is only a matter of seconds to have another try. The fine needle cannot possibly do any damage, though it is possible that it and the injection of 0.5 ml. of fluid into the cavity might stimulate a contraction in a heart which had practically ceased to beat.

The great advantage of the method is that it is certain. The dosage does not depend on or get lost in the peripheral circulation before it reaches the heart, and, if by any chance the result is not, as Mr. Mills says, "dramatic" owing to the heart having ceased to beat, I have in one case found that firm alternating pressure on the thorax apparently initiated enough circulation to take the blood to the brain and make the baby breathe. In such cases milking the umbilical cord would have been useless, and of course all oxygenating methods—such as that mentioned by Dr. David Morris (p. 675)—depend on the existence of quite a reasonable peripheral circulation. I am glad to see that quite a few textbooks now suggest intracardiac injections. The great

difficulty is to get people to use a drug, cardiazol ephedrine, which no textbook mentions but which is much more effective and logical than nikethamide ("coramine"), and still more so than lobeline, which is practically useless in these cases.

May I take this opportunity to correct and apologize for a slip in my previous letter (*Journal*, August 29, p. 510)? I there wrote that you noted (*Journal*, July 25, p. 199) that, while the maternal mortality has been quartered in the last 20 years, the stillbirth rate remains unchanged. What you actually wrote was: "While in the last 20 years the maternal mortality has been quartered, stillbirths and neonatal deaths have shown no comparable decline." The difference is significant, though not sufficiently so to affect the argument or need correction except in a scientific journal. I am sorry it happened.—I am, etc.,

Winsford, Cheshire. W. N. Leak.

Some Complications of Temporal Arteritis

Sir,—Dr. David Kendall's article entitled "Some Complications of Temporal Arteritis" (*Journal*, August 22, p. 418) comments on the occurrence of this disease in arthritic patients.

A woman of 78 years in good health, and who had not previously suffered from arthritis, experienced sudden acute pain in the right hip-joint. The onset of the pain could be timed to within a few minutes, occurring on rising from her chair after lunch. The pain was of such intensity as to suggest a fracture of the neck of the femur. The leg could not be raised from the bed for several weeks. This attack was shortly followed by a similar acute pain in the left hip-joint, and two months later by temporal arteritis affecting both temporal arteries and the occipital branch of the right occipital artery. As the arteritis resolved, the patient rapidly became blind in the right eye, and has only a little vision in the left eye.

I would like to suggest that the lesion in the region of the hip-joints was possibly of the same nature as the inflammatory condition of the other arteries with the same type of onset and resolution.—I am, etc.,

Newport, Mon. K. V. Crowther.

The Age of the Menarche

Sir,—Referring to Drs. Dagmar C. Wilson and Ian Sutherland's memorandum (*Journal*, September 12, p. 607), Stefansson[1] mentions that the girls in the Arctic begin with their menstrual period at an early age, but he points out that they protect themselves against the cold so efficiently that their bodies are as warm as if they were in the Tropics. This is further evidence that climate is not the determining factor, and suggests that clothing must be taken into consideration.—I am, etc.,

Wilpshire, Lancs. Arthur H. Gregson.

References

[1] *The Friendly Arctic*, 1921, London.

Emergency Bed Service

Sir,—Dr. Stephen J. Hadfield reports, in his field survey of general practice (*Journal*, September 26, p. 683), that the Emergency Bed Service has attracted a certain amount of adverse criticism. This is perhaps inevitable, but he adds: "There seems a lot to be said for the principle that if a general practitioner has decided that admission to hospital is urgently necessary then the job of the Emergency Bed Service should be to find a bed and not first to decide whether or not a bed should be found."

The Service is only too well aware of the resentment felt by many practitioners at being asked clinical details by lay staff. It fully recognizes that its job is to find a bed and not to "decide whether or not a bed should be found"—a decision which, as a lay organization, it does not and cannot make. With nothing but a bare diagnosis, however, finding a bed is always a difficult and hopeless task. Deplorable though it may seem to general practitioners, the fact remains that the majority of London hospitals are unwilling to accept a case from the E.B.S. without clinical details. The information requested from the practitioner, which varies with the diagnosis, is that which experience has shown is most often required by the hospitals, and which the doctor himself presumably gives if he applies direct to the hospital.

A further reason for requiring clinical details is that, on occasions when difficulty is experienced in finding a bed, the case is submitted to a doctor representing the regional boards. This doctor cannot assess the case and give instructions to the Service without adequate information.—I am, etc.,

G. F. Abercrombie,
Chairman, Emergency Bed Service Committee,
King Edward's Hospital Fund for London.

London, E.C.2.

The Method of Execution

Sir,—I am amazed at the complacency of chaplains and others not about to be hanged regarding our present method of execution. It is defended because only 9 to 12 seconds are said to elapse between the procession from the condemned cell and the pulling of the scaffold lever, as against the minute which would elapse before unconsciousness supervened upon the first inhalation of carbon monoxide gas.

It is a well-known fact that the first effect of carbon monoxide poisoning is a blunting of the emotions, while in any case there would be no need for a staged execution, and the fatal gas, being odourless, could be introduced without pomp and circumstance, after the expiration of the customary period for grace.

In contrast, let any person of imagination picture himself taking the horrid drop with its awful culmination, after the expiration of those few seconds. Would anyone, Sir, not prefer for himself almost any other method?

As a doctor, I would not hesitate to administer euthanasia to any unfortunate so condemned to execution, and I would be infinitely grateful to anyone who would administer to me any hypodermic or intravenous injection to save me from the horror of our present barbarous method.—I am, etc.,

South Molton, N. Devon. R. A. Nash.

Sir,—May I most emphatically dissociate myself from the activities of the B.M.A. representatives who have been discussing improved methods of killing persons convicted of murder?

May I point out that our activities should only be concerned with life and its preservation and that the B.M.A. has no place in discussions on taking life?

Finally, may I please be permitted to suggest that, if as an Association we are consulted, we should in all humility recognize that there are no circumstances which absolve us individually or collectively from the ancient Commandment "Thou shalt not kill"?—I am, etc.,

Gateshead, 8. K. T. Brown.

St. Luke's Day

Sir,—The small international society of which Luke, the physician, was a foundation member is now world-wide. On October 18 its many thousands of members recall with gratitude his life and work. How much poorer the world would be had Luke never lived and laboured. We of the medical profession may well take our part in commemorating with gratitude his life and work, and join in the hope and prayer that by the prescriptions he has left to us all the diseases of the world's mind and soul may be cured.

Shakespeare's Macbeth demands of his physician: "Canst thou not minister to a mind diseased?" It is in this branch of medicine that Luke excelled. Though it is rather as a writer than a physician that he has gained fame, his writings entitle him to a high place of honour among the world's psychiatrists. How many minds, sick with foolishness and selfishness, have found relief in studying his textbook.—I am, etc.,

Banstead, Surrey. Herbert Caiger.

Obituary

J. F. CARTER BRAINE, M.D., F.R.C.S., F.F.R.

Dr. J. F. Carter Braine, director of the radiotherapeutic department of Guy's Hospital, died at his home at Speldhurst, Kent, on September 20 at the age of 60.

Born on January 21, 1893, John Francis Carter Braine received his medical education at Guy's Hospital, qualifying M.R.C.S., L.R.C.P. in 1916. After qualification he entered the R.A.M.C. and served in France and East Africa in the first world war. On demobilization he returned to Guy's as demonstrator of anatomy, and later was appointed Griffiths demonstrator in the department of pathology, a post he held for some years, during which time he took the Durham M.D. in 1925, winning a gold medal for his thesis. He had previously graduated M.B., B.S. at Durham in 1920; in 1921 he took the D.P.H. of the English Royal Colleges; and in 1922 he obtained the diploma of F.R.C.S. It is interesting to recall that both his father and grandfather were Fellows of the Royal College of Surgeons of England.

Always attracted to radiotherapy, Carter Braine was chief assistant in the old actinotherapy department at Guy's Hospital from the mid-twenties, with the late Colonel W. Langmuir Watt, who died two months ago, as an assistant surgeon. In 1925 Carter Braine was appointed surgeon to the radiotherapy department of the hospital, and in due time he acquired a wealth of experience in this field of treatment, working, as he did, unceasingly to improve techniques and results, so that his selection in 1939 as director of the department was an obvious choice. In the same year he was elected a Fellow of the Faculty of Radiologists.

During the last war he acted as adviser in radiotherapy to the group officer in the Guy's sector, and when, in 1941, the Guy's U.S.A. Hospital was opened at the Wildernesse, near Sevenoaks, he helped to create in hutted accommodation a radiotherapy department which was in use until the end of the war, when it was transferred to Pembury Hospital. From 1941 to the end of hostilities it is estimated that Carter Braine travelled by road each year in black-out conditions at least 20,000 miles between Tunbridge Wells, Pembury, the Wildernesse, and London in the performance of his duties. The strain such calls made on his frame, robust though it was, must have provided fertile ground on which the seeds of his fatal illness were sown. His ability as an organizer showed itself most clearly during the war years; an inspiration to his staff, Carter Braine's enthusiasm and encouragement produced a remarkable loyalty in those under him.

He was civil consultant in radiotherapy to the Royal Navy, and was formerly surgeon in charge of the radium department at King Edward VII Hospital, Windsor, and radiotherapist to the West London Hospital. He was a foundation member of the Society of Radiotherapists of Great Britain and Ireland (now part of the Faculty of Radiologists). A member of the official British delegation to the fifth International Congress of Radiology held at Chicago in 1936, he was also assistant treasurer and a member of the board of management of the sixth International Congress of Radiology, held in London in 1950. Carter Braine had wide interests outside his profession. His love of the countryside was shown in the careful husbandry of his farm. Art and architecture, too, appealed to him; and he was a competent photographer, as the fine clinical photographs and slides used in his department could testify. His unpretentious manner, his modesty, and his sympathy and understanding inspired the affection of colleagues and patients, to whom he was the beloved physician. A keen sense of humour and an even temperament ensured him many friends, who, with his widow and daughter, have suffered a very severe loss by his death.

L. A. ROWDEN, M.B., D.Sc., F.F.R.

The death occurred at Harrogate on August 16 of Dr. L. A. Rowden, a former president of the British Institute of Radiology, at the age of 82.

Leonard Alfred Rowden was born at Newcastle-upon-Tyne on March 23, 1871, and was educated at Lenzie Academy and at Glasgow and London universities, graduating M.B., C.M., with a commendation, at Glasgow in 1892. After graduation he was in general practice at Thirsk and Morley before finally setting up his plate on his own account at Leeds. The discovery of *x* rays by Röntgen in 1895 saw the birth of radiology, and it was to this branch of medicine that Dr. Rowden devoted most of his working life. He became one of the pioneers in the use of *x* rays, starting before the turn of the century, when the first plant at the General Infirmary at Leeds was installed at a cost of £5 15s., and he later fitted up his own electrical laboratory, one of the earliest of its kind in this country. In 1908 he was appointed director of the *x*-ray department at the General Infirmary at Leeds, and in 1910 he became radiologist to the Hospital for Women and Children, holding both positions until 1917. From 1910 to 1919 he was radiologist to the Leeds Public Dispensary, and he was also honorary consulting radiologist to Dewsbury General Infirmary. In the first world war he served in the R.A.M.C. from 1915 to 1919 at home and in France. After the war he returned to Leeds, where he remained until his last illness. He retired from practice in 1948. The ambulance movement in Yorkshire also received his support. He was examiner and instructor in the ambulance department of the old London and North-Eastern Railway, examiner for the St. John Ambulance Association, and he gave the Rowden challenge shield for yearly competition by the ambulance teams in Leeds workshops and factories.

Dr. Rowden was a past president of the British Institute of Radiology, the Society of Radiographers, and the Leeds and West Riding Medico-Chirurgical Society. A member of the British Medical Association for some forty years, he acted as one of the secretaries of the Section of Radiology and Medical Electricity at the London Annual Meeting of the Association in 1910, and when the B.M.A. met at Glasgow in 1922 he was president of the Section of Radiology. He also represented his constituency at eight Annual Representative Meetings from 1918 to 1927. In 1939 the honorary degree of D.Sc. was conferred upon him by the University of Leeds, and when the British Association of Radiologists and the Society of Radiotherapists of Great Britain and Ireland amalgamated as the Faculty of Radiologists in 1940 he became a Fellow of the Faculty. Dr. Rowden was always ready to help any young man who showed interest in *x*-ray work. He had a fine collection of positive prints and films, and nothing pleased him

more than to be able to show them to visitors. He was not a prolific writer, but his contributions to *A Descriptive Atlas of Radiographs* outnumbered all others. Latterly he was particularly interested in pelvimetry. He was one of the few pioneers who escaped injury through radiation. A widower, he leaves six sons and two daughters. His eldest son, Dr. W. H. Rowden, is in practice at Leeds.

The *British Medical Journal* has lost an old and valued contributor by the death, which took place in Edinburgh on September 27, of Philip Francis Sulley, London editor of the *Sheffield Daily Telegraph* and Parliamentary Lobby correspondent. For many years—indeed, the beginning of the association does not seem to be discoverable from the office records, but it may well have been 30 years or more—right down to the Parliamentary recess last summer, Sulley contributed Medical Notes in Parliament and generally acted as watchdog for the *British Medical Journal* over the proceedings at Westminster. This was, of course, only one of his extra-activities; his main job was to interpret Parliamentary doings to Yorkshire in the form of a London Letter; but he did it, as he did everything else, with great care, good judgment, and a profound knowledge of Parliamentary procedure, personalities, and affairs. He had also, which was very important for such work, a capacity for compression, and could condense into a few lines a statement or fragment of discussion when an amateur would have straddled across a page.

Sulley, who was in his seventieth year, was a Scotsman who, having passed through St. Andrews University, came south to make his reputation. He began his Parliamentary journalistic work about 1906, when the Campbell-Bannerman Government came into power. He had been chairman of the Press Gallery, and twice chairman of the Lobby. He was indeed the "father" of the Lobby, and was much esteemed by his colleagues as well as by Parliamentarians and others as a political journalist, able to express himself with frankness and wit, and always keen on safeguarding the highest standards of journalism. A year or two ago he had a severe illness for which he underwent an operation, and, although he returned to his accustomed haunts at Westminster, he was, as one of his colleagues expressed it, "rather like a winged bird." Proceedings in Parliament cover all human interests, and to dissect from the daily question times and from the debates in the House and its committees matters of interest in health and medicine called for an alert mind and no uncommon labour. But Sulley did his job admirably and his practised pen will be greatly missed.

We record with regret the death on September 7 of Miss Winifred Linton, honorary F.C.S.P., formerly head of the department for remedial exercises at the Brompton Hospital.

Sir Geoffrey Marshall writes: The death of Winifred Linton brings to an end a life of unremitting work which formed one of the stepping-stones for the spectacular rise of chest surgery and medicine in our time. She was trained as a nurse at St. Bartholomew's Hospital, and served as such with the Army in the Dardanelles in the first world war. After her return home she took up physiotherapy, and began her association with the Brompton Chest Hospital, where she developed and was head of the department for remedial exercises. There she worked first with the late Mr. J. E. H. Roberts, and then with the late Mr. Tudor Edwards, whose brilliant team-work set new standards in surgery which have since become standardized in every civilized country. Miss Linton was the most modest and self-regardless of women; but she was a genius. If the disability of a patient was explained to her, she would devise exercises and postural procedures which would reduce the disability to a minimum, and she would gain the confidence and eager co-operation of patients of all ages and of every rank of society. A patient about to undergo a major operation, such as the removal of the whole of one lung, would be taught beforehand how to breathe and cough using the other lung only. She was equally successful in dealing with patients suffering from chronic disease, showing them how to overcome the shortness of breath associated with such conditions as asthma and chronic bronchitis. She trained many other physiotherapists, and her pupils have become heads of departments in hospitals here and abroad. She was a woman of small stature and of no special distinction in appearance; but her zest and her complete devotion to the relief of her patient carried instant conviction and made her respected and beloved. She had a delicious sense of humour, and she was a dear. Her work lives on and expands.

Dr. Homer Fordyce Swift, a former member of the Rockefeller Institute for Medical Research, died in New York on September 24 aged 72. He graduated M.D. New York and Bellevue Medical College in 1906. He was assistant pathologist and dermatologist at Bellevue from 1908 to 1910, and then became associated with the staff of the Rockefeller Institute. In 1914 he was appointed associate professor of medicine at Columbia University, and three years later obtained a similar appointment at Cornell Medical College. In 1912, with A. W. M. Ellis, he introduced the autoserosalvarsan method of treatment of neurosyphilis (the "Swift–Ellis treatment"). During service with the Allied Armies in France in 1918 he collaborated in the production of an important report on trench fever for the American Red Cross Society's Commission. He returned to the Rockefeller Institute as an associate member in 1918, becoming a full member three years later. He was made a physician to the Rockefeller Institute for Medical Research in 1936; he was a member of the Council of New York University from 1942 to 1946, and consultant to the Secretary of War during the same period.

Medico-Legal

CLAIM AGAINST DOCTORS FAILS

After a five-day hearing, Mr. Justice Finnemore dismissed on October 9 at Birmingham Assizes a claim for damages for personal injuries by a man who contended that the injuries resulted from a supposition that he had venereal disease when he was actually suffering from cancer. Later amputation of the penis had been performed.

Daniel Edwin Hulse, 36, of Oldbury, Worcs, alleged negligence by Dr. C. J. Wilson, resident surgical officer, and Mr. J. H. Kirkham, stipendiary surgeon, both of the West Bromwich and District General Hospital, and also against the Birmingham Regional Hospital Board, who administer the hospital.

The defence was a denial of negligence.

Mr. E. Ryder Richardson, Q.C. (for Hulse), opening the case, said that in 1948 Hulse underwent a number of tests for venereal disease at a Birmingham hospital. The tests proved negative. He was also seen at West Bromwich Hospital. The pain increased, and on January 1, 1949, Hulse entered the West Bromwich Hospital, where they still thought he was suffering from the disease. An operation was performed and he was sent home three days later. In March Hulse saw another surgeon, who began deep *x*-ray treatment for cancer, but on July 25 an amputation operation was performed. Mr. Ryder Richardson alleged that the doctors had wrongly diagnosed a disease as the source of the trouble, instead of cancer, and Hulse was claiming damages for personal injuries.

Giving evidence, Hulse said he had nineteen operations altogether, including several plastic surgery operations.

His last operation was on May 27 this year, and he still suffered from pains in the lower abdomen and had spells of giddiness.

Dr. H. Barrada, Hulse's doctor, said he thought Hulse was fit enough to do clerical work, but unable to carry out many forms of manual work, to which he had been accustomed.

Mr. M. Harmer, of London, said that if the diagnosis of Hulse's trouble had been made earlier he might eventually have had to have only a partial amputation instead of a total one. The subsequent extensive plastic operations would also probably have been found unnecessary, because a man with a partial amputation was to a large extent able to carry on a normal life. He thought that a man who had only a partial amputation could work better, and there was less risk of infection, than if he had a total amputation. In his opinion, if Hulse had been treated in January, 1949, three months would have been saved, and the man might have escaped with a partial amputation, but he agreed in cross-examination that in 1948 a time lag of about six months before admission to hospital was not unreasonable for a minor operation.

Dr. William Henry Bond, consultant radiotherapist to United Birmingham Hospitals, said that the cancer did not respond to radiotherapy, but it would probably have been resistant to radiotherapy even if it had been diagnosed earlier. Having regard to Hulse's age, cancer was the least probable cause of this condition, and he thought Dr. Wilson had acted correctly under the circumstances.

Professor B. W. Windeyer, director of radiotherapy at Middlesex Hospital, London, said there was no reason why Dr. Wilson should suspect cancer when he first examined Hulse. In a man of Hulse's age such a cancer was extremely rare.

Professor F. A. Stammers, professor of surgery at Birmingham University, said he considered that Mr. Kirkham's actions after the minor operation in January, 1949, were quite reasonable.

Mr. Kirkham, giving evidence, said that he did not see Hulse at all until he performed a minor operation on him in January, 1953. The operation disclosed an ulcer, which might have been due to one of three causes. He considered cancer to be the least likely one, particularly in view of Hulse's age.

Mr. Vaughan (for Dr. Wilson and the regional hospital board), in his closing speech said there was a waiting-list of six to nine months in 1948 for operations at the hospital. There was nothing to induce any reasonably minded doctor to think that there was anything in Mr. Hulse's case requiring a higher priority than for the first operation—a very minor one.

On behalf of Mr. Kirkham, Mr. A. P. Marshall, Q.C., said that Mr. Kirkham had acted reasonably and properly in delaying the serious surgical cancer test until after the possibilities of inflammation and syphilis had been disproved. There was no evidence of negligence on Mr. Kirkham's part.

The judge, dismissing the claim, said that he found no evidence of any negligence against any of the defendants. There was no reason for Dr. Wilson to suspect cancer in the early stages, and at that time there was no reason for the case to be treated as one of urgency. Mr. Hulse had not been told to go back to the hospital if the pain became worse, but there was nothing to stop him going to his doctor, or to the hospital, if this happened. "I don't think you can blame the doctor for not saying 'Please come back' to him," said the judge.

Referring to the question of whether Mr. Kirkham should have diagnosed cancer when he discovered an ulcer during an operation, the judge commented: "The dangers of making a diagnosis too quickly are just as great as making a diagnosis too slowly. I don't think anyone can complain that Mr. Kirkham did not diagnose cancer straight away.

"I think the real gravity of the case against Mr. Kirkham was that he delayed too long satisfying himself beyond doubt that venereal disease was not the cause, and that he should have turned to the only other alternative—cancer. Such a cancer is extremely rare: in a young man of about 30 it is rarer still. Until you get rid of the other more likely causes you would not expect it was cancer in a man of this age." In any event, if Mr. Kirkham had at once diagnosed cancer no one could say that Mr. Hulse could have avoided an amputation altogether.

Messrs. Hempsons acted on behalf of the Medical Defence Union for Mr. Kirkham.

INFECTIOUS DISEASES AND VITAL STATISTICS

Summary for British Isles for week ending **September 26** (No. 38) and corresponding week 1952.

Figures of *cases* are for the countries shown and London administrative county. Figures of *deaths* and *births* are for the 160 great towns in England and Wales (London included), London administrative county, the 17 principal towns in Scotland, the 10 principal towns in Northern Ireland, and the 14 principal towns in Eire.

A blank space denotes disease not notifiable or no return available.

The table is based on information supplied by the Registrars-General of England and Wales, Scotland, N. Ireland, and Eire, the Ministry of Health and Local Government of N. Ireland, and the Department of Health of Eire.

CASES in Countries and London	1953 Eng. & Wales	1953 Lond.	1953 Scot.	1953 N. Ire.	1953 Eire	1952 Eng. & Wales	1952 Lond.	1952 Scot.	1952 N. Ire.	1952 Eire
Diphtheria	29	2	3			21	2	12	1	
Dysentery	263	45	88	4	4	103	21	96	3	1
Encephalitis, acute	5	0	0	1		4	0	0	0	
Enteric fever:										
Typhoid	6	2	0	1	2	5	0	0	0	2
Paratyphoid	13	6	2(B)	0	1(B)	33	1	2(B)	0	0
Food-poisoning	274	20		2		160	20		0	
Infective enteritis or diarrhoea under 2 years				16	42				20	41
Measles*	492	13	76	15	32	2,590	272	59	25	61
Meningococcal infection	33	1	15	0		30	3	6	1	
Ophthalmia neonatorum	28	2	3	0		31	5	6	0	
Pneumonia†	245	13	122	1	1	261	12	165	5	
Poliomyelitis, acute:										
Paralytic	119	8	21	28	12	127	10	14	9	4
Non-paralytic	99	7				61	5			
Puerperal fever§	225	37	25	0		253	38	5	1	2
Scarlet fever	786	42	176	32	39	852	60	242	17	43
Tuberculosis:										
Respiratory			110	15				133	28	
Non-respiratory			24	3				27	9	
Whooping-cough	2,771	190	230	27	26	1,366	77	87	19	123

DEATHS in Great Towns	1953 Eng. & Wales	1953 Lond.	1953 Scot.	1953 N. Ire.	1953 Eire	1952 Eng. & Wales	1952 Lond	1952 Scot.	1952 N. Ire	1952 Eire
Diphtheria	0	0	0	0	0	0	0		0	0
Dysentery	0	0		0	0	0	0		0	
Encephalitis, acute		0					1			0
Enteric fever	0	0	0	0		0	0		0	
Infective enteritis or diarrhoea under 2 years	10	1	2	1	3	6	0	1	1	4
Influenza	4	0	0	0	0	5	1		0	0
Measles		1	0	0	0		0		0	0
Meningococcal infection		0	0				0			
Pneumonia	120	15	16	4	5	133	20			4
Poliomyelitis, acute	7	0			0	9	0			0
Scarlet fever		0	0	0	0		0		0	0
Tuberculosis:										
Respiratory	78	9	14	3	4	95	12	14	1	6
Non-respiratory		0	4	0	1		0	2	0	1
Whooping-cough	1	0	0	0	0	1	0		2	0
Deaths 0–1 year	223	24	24	3	19	191	17	25	10	13
Deaths (excluding stillbirths)	4,318	566	502	87	141	4,386	640	543	96	147
LIVE BIRTHS	7,494	1098	818	217	349	7,368	1128	798	198	361
STILLBIRTHS	185	27	22			188	23	21		

* Measles not notifiable in Scotland, whence returns are approximate.
† Includes primary and influenzal pneumonia.
§ Includes puerperal pyrexia.

Vital Statistics

Poliomyelitis

Poliomyelitis notifications (uncorrected) in the week ending October 3 (39th week of the year) were as follows: paralytic, 79 (119); non-paralytic, 78 (99); total, 157 (218). This is a decrease of 61 compared with the previous week, the figures for which are in parentheses. The reduction is satisfactory, and there is no doubt that the drop to the winter levels of the disease is under way.

Cancer Statistics

The Royal Cancer Hospital, London, has issued the first of a series of reports giving detailed statistics on the type of cancer and results of treatment in patients admitted to it. It is entitled *Clinical Statistical Report* (Number 1), and covers the years 1945–9. The report includes figures showing the yearly survival and recurrence rates of all new patients with cancer, classified by site, age, sex, clinical stage, and histological type. In the words of its introduction, "It is intended for those who are seriously interested in studying the position as it is to-day rather than in advocating one particular treatment method or supporting some already entrenched belief." The authors are Mr. A. H. Hunt, Dr. J. B. Harman, Mr. M. H. Harmer, Mr. P. M. Payne, B.Sc., Professor D. W. Smithers, and Mr. K. M. Turner, B.Sc.

Infectious Diseases

The largest variations in the number of notifications of infectious diseases in England and Wales during the week ending September 26 were decreases of 784 for whooping-cough, from 3,555 to 2,711, and 116 for measles, from 608 to 492, and increases of 153 for scarlet fever, from 633 to 786, and 61 for dysentery, from 202 to 263.

The incidence of scarlet fever rose slightly in most regions of the country. The largest local variation was an increase of 38 in the number of notifications in Lancashire, from 117 to 155. The largest decreases in the number of notifications of whooping-cough were 103 in Yorkshire West Riding, from 317 to 214; 91 in London, from 281 to 190, 77 in Essex, from 223 to 146, and 73 in Glamorganshire, from 148 to 75. A small decline in the incidence of measles was recorded in most areas, but no large fluctuations were reported. 29 cases of diphtheria were notified; these were 4 more than in the preceding week and the largest total for 28 weeks. The largest local fluctuation in diphtheria was a decrease in Durham from 4 to 0. 6 of the 13 cases of paratyphoid fever and 2 of the 6 cases of typhoid fever were notified in London.

218 cases of acute poliomyelitis were notified during the week. These were 4 fewer for paralytic and 5 fewer for non-paralytic cases than in the preceding week. The largest returns were Warwickshire 29 (Coventry C.B. 11, Birmingham C.B. 7, Shipton-on-Stour R.D. 3), Lancashire 20 (Preston C.B. 5, Blackpool C.B. 4, Liverpool C.B. 4), London 15 (Lewisham 3, St. Pancras 3), Southampton County 12 (Bournemouth C.B. 3), Yorkshire West Riding 12 (Sheffield C.B. 3), and Northamptonshire 11 (Kettering M.B. 8).

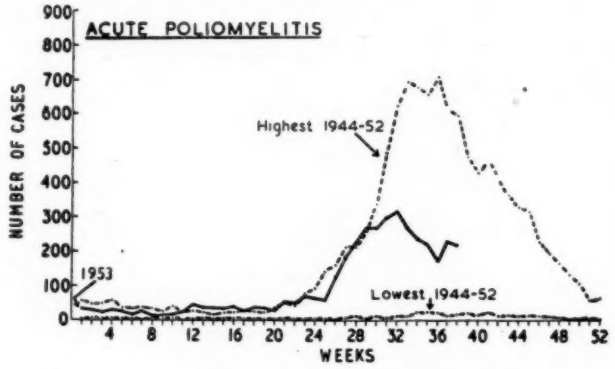
The largest centres of dysentery during the week were London 45 (Southwark 12, Chelsea 9), Lancashire 30 (Liverpool C.B. 7), Yorkshire West Riding 22 (Bradford C.B. 8), Durham 18 (Stanley U.D. 9), Middlesex 17, Kent 14 (Maidstone M.B. 9), Worcestershire 12 (Bromsgrove U.D. 12), Surrey 10, and Yorkshire North Riding 10 (Middlesbrough C.B. 10).

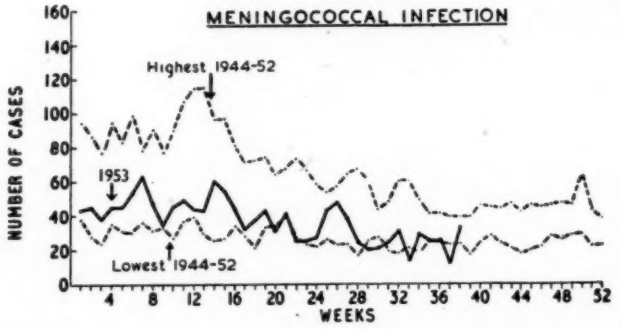
Week Ending October 3

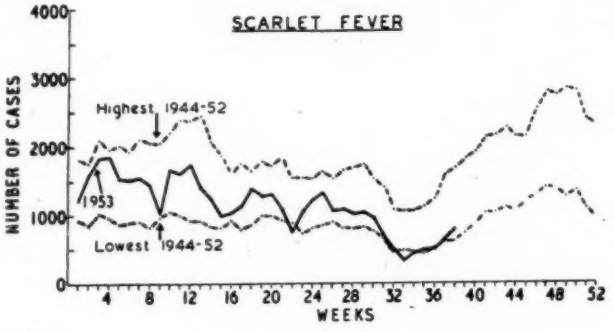
The notifications of infectious diseases in England and Wales during the week included scarlet fever 992, whooping-cough 2,292, diphtheria 24, measles 621, dysentery 212, paratyphoid fever 16, and typhoid fever 10.

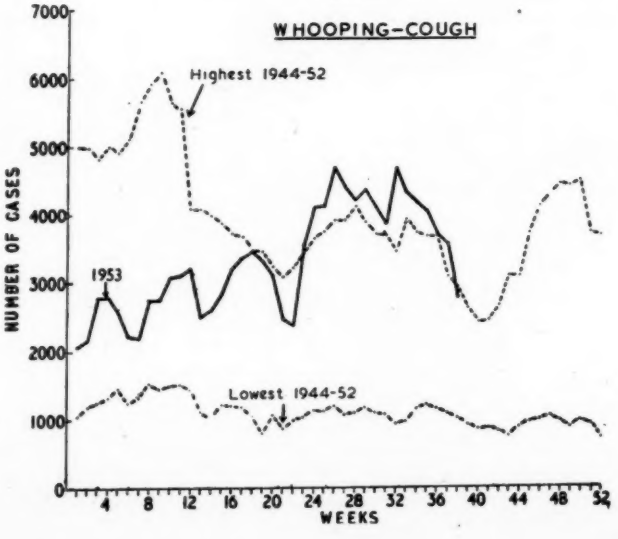
Graphs of Infectious Diseases

The graphs below show the uncorrected numbers of cases of certain diseases notified weekly in England and Wales. Highest and lowest figures reported in each week during the nine years 1944–52 are shown thus - - - - - - -, the figures for 1953 thus ————. Except for the curves showing notifications in 1953, the graphs were prepared at the Department of Medical Statistics and Epidemiology, London School of Hygiene and Tropical Medicine.









Medical News

Postgraduate Travelling Fellowships.—The British Postgraduate Medical Federation (University of London) is prepared to offer a limited number of Postgraduate Travelling Fellowships, tenable for one year, to registered medical and dental practitioners who are in the course of training as specialists in one of the preclinical or clinical branches of medicine, surgery, or dentistry, at one of the medical or dental schools or teaching hospitals and Postgraduate Institutes of the University of London. Further information about these Fellowships will be found in the advertisement columns.

The Pathologist in Court.—Speaking from the chair of the Medical Protection Society, to which he was unanimously re-elected at its annual meeting on October 7, Sir Ernest Rock Carling referred to the tendency in some coroners courts for pathologists to express opinions on clinical matters. The pathologist had the remarkable advantage of being wise after the event and of entire exemption from the responsibility of having decided what the treatment of the deceased person should have been. He considered that in expressing such opinions the pathologist or morbid anatomist was stepping outside his proper province, and in his view coroners should not in future attach the importance some of them seemed to do to the clinical opinions of pathologists. Sir Ernest Rock Carling went on to say that the Society had been anxious about the attitude taken by some hospital authorities to complaints against members of their staff. In repudiating liability on its own account one authority had gone so far as to present the plaintiff with a series of points on which it was alleged a member of the staff had been negligent. One regional board had told its staff that they would individually be held responsible for any accident connected with blood transfusion which might occur at the hands of registrars, house-surgeons, and others looking after their patients. On these matters representatives of the defence societies and the British Medical Association had been conducting negotiations with the Ministry of Health, and there were signs that a different attitude between the employers and the employed staff in this respect might obtain in the future. He also referred to the tendency to increase the amount of damages awarded against doctors.

Charing Cross Hospital Medical School Dinner.—A record number of past and present students attended the annual dinner at the Savoy Hotel, London, on October 10, under the chairmanship of Lord Inman. The occasion was marked by farewell presentations to Mr. Norman Lake, the senior surgeon, and Professor H. W. C. Vines, head of the pathology department and formerly Dean, who are retiring under the age limit. Responding to the toast of "The Medical School" proposed by Dr. C. M. Scott, the Dean, Dr. E. C. Warner, reviewed the School's progress. This done, he turned to the main business of the evening. A fund had been opened to commemorate the many years' service by Mr. Lake and Professor Vines, and £720 had been collected. The bulk of the money would go to establish a Norman Lake prize for clinical surgery and to augment the Vines prize in pathology. Both Mr. Lake and Professor Vines, he was glad to say, would continue to work for the hospital, the former as chairman of the hospital research committee and, for a time, as director of surgical studies, the latter on the board of governors and the school council. Mr. David Trevor, to whom it fell to propose their healths, nicely blended reminiscence with appreciation. Mr. Lake had been on the staff for 34 years and senior surgeon for 20 years—surely a record. He would be remembered as a general surgeon in the widest sense of the word. Professor Vines, who had come to the hospital in 1928, was distinguished as an educationalist and an organizer. Above all, he was the force behind the planning of the new hospital at Harrow. The chairman then presented a tape recorder to Mr. Lake, on which the proceedings of the evening were being registered, and a cocktail cabinet to Professor Vines. Mr. Lake, whose speech will long be remembered, spoke of his great gratitude to the hospital, which had afforded him a "lifetime of most absorbingly interesting work." Professor Vines asked his hearers to remember that the life of an institution depended, not on its buildings, but on the enthusiasm and good will of its alumni. He predicted a rosy future for the Charing Cross School. These speeches were greeted with enthusiasm, and the evening ended with a vote of thanks to Lord Inman by Mr. Arthur Gray.

King's College Hospital Medical School Dinner.—The annual dinner of past and present students was held at the Dorchester Hotel, London, on October 3, with Mr. V. E. Negus in the chair. The toast of the chairman was proposed by Mr. W. I. Daggett, and Mr. V. E. Negus replied on behalf of himself and the guests, and gave the toast of the medical school. The wit of the speakers fittingly matched the brevity of their speeches, to the evident delight of the diners. It was announced that next year would be the 125th anniversary of the medical school, when it is hoped that a specially large number of past students will be able to take part in the celebrations.

The London Hospital Old Students' Dinner was held at the Trocadero Hotel, London, on October 8. Mr. George Neligan, from the chair, proposed the toast of "The Hospital and Medical School," which was replied to by Dr. A. E. Clark-Kennedy, who is retiring as Dean of the Medical College. Dr. A. G. Maitland-Jones proposed the chairman's health. Among the guests were Professor A. D. Gardner, Sir Lionel Whitby, Lieutenant-General Sir Frederick Harris, Surgeon Vice-Admiral Sir A. Ingleby-Mackenzie, Air Vice-Marshal J. M. Kilpatrick, and Sir James Spence, who had earlier in the day delivered the Schorstein Lecture on "The Varieties of Clinical Experience."

Charles Best Institute.—The new Charles H. Best Institute, at the University of Toronto, was officially opened on September 15, when Sir Henry Dale, O.M., turned a golden key in the door of the new building. The Institute will be used by the Department of Physiology for teaching purposes, research, and by the associated Banting and Best Department of Medical Research. At a special convocation held before the opening, the University presented honorary degrees to Dr. Detlev W. Bronk, the new Director of the Rockefeller Institute for Medical Research; to Dr. B. A. Houssay, of Argentina; to Dr. E. P. Joslin, of Boston; to Dr. W. G. Penfield, of Montreal; and to Sir Lionel Whitby, the Vice-Chancellor of the University of Cambridge. Convocation addresses were given by Sir Henry Dale and by Sir Lionel Whitby.

Italian Visit by R.S.M. Delegation.—At the invitation of the president of the Lombardy Region of the Italian Red Cross a small delegation of Fellows of the Royal Society of Medicine have been lecturing in Northern Italy. The party was led by Sir Gordon Gordon-Taylor, and included Sir Clement Price Thomas, Mr. L. R. Broster, Professor L. J. Witts, Dr. H. M. Sinclair, and Mr. R. T. Hewitt, secretary of the Royal Society of Medicine. Lectures were given at the Universities of Milan, Pavia, and Padua.

Opinion on Crime.—The Nuffield Foundation has invited Lord Pakenham, a Fellow of Christ Church, Oxford, and formerly Minister of Civil Aviation and First Lord of the Admiralty, to undertake a critical appraisal of current views of the causes of crime. He will be assisted by a small group of assessors, some of whom are medical. They are Dr. Desmond Curran, psychiatrist at St. George's Hospital, and Dr. T. C. N. Gibbens, lecturer in forensic psychiatry at the Institute of Psychiatry. The non-medical assessors include Dr. M. Grünhut, reader in criminology at Oxford, Mr. Frank Milton, a Metropolitan magistrate, and Mr. Leslie Farrer-Brown, secretary of the Nuffield Foundation.

Grenfell Association Christmas Cards.—Once again the Grenfell Association is offering Christmas cards for sale, all proceeds of which will be devoted to the maintenance and furtherance of the Grenfell Mission's medical work in Labrador and Northern Newfoundland. An illustrated leaflet giving details of cards available (ranging in price from 3½d. to 1s. 2d.) may be obtained from the association office, 66, Victoria Street, London, S.W.1, on receipt of 1½d.

Electroencephalographic Society.—At the annual general meeting of the Electroencephalographic Society, held at the National Hospital, Queen Square, on October 3, Dr. Denis Hill (Institute of Psychiatry), and Dr. Geoffrey Walsh (Edinburgh) were elected to the council to fill vacancies caused by retirement. The general meeting was followed by a series of papers. Summaries may be obtained from the honorary secretary, Dr. D. A. Pond, Maudsley Hospital, London, S.E.5.

The Annual Report of the Board of Medical Auxiliaries for 1952–3 records that the Association of Operating Theatre Technicians has been recognized as a qualifying body. A register of operating theatre technicians is to be published in due course. The British Dietetic Association has withdrawn its membership.

The Births and Deaths Registration Act, 1953, came into force on September 30. It simply consolidates, with a few minor amendments, previous legislation on the registration of births and deaths in England and Wales.

Sir James Learmonth, regius professor of clinical surgery at Edinburgh, has been appointed Sir Arthur Sims Commonwealth Travelling Professor for 1954 by the Royal College of Surgeons of England.

Sir Harry Platt will leave on October 24 for Cyprus and Israel for a two weeks' lecture tour under the aegis of the British Council. He will spend the first week in Cyprus, giving a number of formal lectures and clinical demonstrations, and the second week in Israel, where he will lecture, hold clinics, and operate in Tel-Aviv, Haifa, and Jerusalem.

COMING EVENTS

Cambridge University Courses for General Practitioners.—A series of one-day symposia for general practitioners will be held on Saturdays at Addenbrooke's Hospital during the academic year. The following programme has been arranged: October 24, "Tuberculosis and Other Diseases of the Chest"; November 14, "Disorders of the Blood"; December 12, "Dermatology"; January 9, "Obstetrics and Gynaecology"; February 6, "Infectious Diseases"; March 6, "Cardiovascular Diseases"; April 3, "Orthopaedics, Treatment of Injuries and Physical Medicine"; May 1, "Cancer"; May 29, "Paediatrics"; June 26, "Nervous Diseases"; July 24, "Diseases of the Ear, Nose, and Throat; Diseases of the Eye." Further information may be obtained from the secretary, the Medical School, Tennis Court Road, Cambridge.

University College Hospital Medical School.—The annual dinner of past and present students will be held at the Savoy Hotel, London, W.C.2, on October 30 at 7.30 for 8 p.m. Dr. Kenneth H. Stokes will be in the chair. Tickets (price £1 7s. 6d.) may be obtained from the dinner secretary, University College Hospital Medical School, London, W.C.1. On October 29 and 30 there will be the usual postgraduate demonstrations at the Medical School. These include a symposium on the treatment of severe hypertension.

27th General Hospital Reunion.—The annual dinner will be held at the Eccleston Hotel (near Victoria) on October 31 at 6.15 for 7 p.m. Tickets £1, inclusive of wine; informal dress. Those wishing to attend should communicate with Dr. L. S. Potter at B.M.A. House, Tavistock Square, London, W.C.1.

The British Occupational Hygiene Society.—The first conference of the society will be held on November 2 at 11 a.m. at the London School of Hygiene and Tropical Medicine, Keppel Street, London, W.C.1. The Minister of Labour, Sir Walter Monckton, will open the conference, and Dr. T. Bedford will deliver the presidential address. The main subjects for discussion will be the measurement of the worker's environment, chemical toxicity, and occupational hygiene and the industrial designer.

SOCIETIES AND LECTURES

A fee is charged or a ticket is required for attending lectures marked ●. Applications should be made first to the institution concerned.

Friday, October 16

ROYAL EYE HOSPITAL.—5.30 p.m., Dr. A. A. Guild: Ocular Manifestations of Neurosyphilis.

Monday, October 19

●HUNTERIAN SOCIETY.—At Talbot Restaurant, 64, London Wall, E.C., 7 for 7.30 p.m., dinner meeting. Presidential Address by Sir Heneage Ogilvie: Life is Like That.

INSTITUTE OF NEUROLOGY.—5 p.m., Dr. Robert Aird (U.S.A.): Recent Work on Epilepsy.

INSTITUTE OF PSYCHIATRY.—5.30 p.m., Dr. E. Stengel: lecture demonstration.

LONDON UNIVERSITY.—At Guy's Hospital Medical School, 5 p.m., Professor William Montagna (Brown University): The Architecture, Growth, and Differentiation of Skin.

●POSTGRADUATE MEDICAL SCHOOL OF LONDON.—4 p.m., Dr. B. G. C. Ackner: Aches and Pains of Psychogenic Origin.

Tuesday, October 20

BRITISH POSTGRADUATE MEDICAL FEDERATION.—At London School of Hygiene and Tropical Medicine, 5.30 p.m., Professor J. H. Burn, F.R.S.: Acetylcholine and Cholinesterase in Heart Muscle.

INSTITUTE OF DERMATOLOGY.—5.30 p.m., Dr. D. I. Williams: Erythrodermias.

ROYAL EYE HOSPITAL.—5 p.m., Dr. R. D. Lawrence: Diabetes and the Eye.

ST. MARY'S HOSPITAL MEDICAL SCHOOL.—At Wright-Fleming Theatre, 5 p.m., Mr. A. M. Claye: Sexual Disorders.

SOCIETY FOR THE STUDY OF ADDICTION.—At Medical Society of London, 8 p.m., quarterly meeting. Presidential Address by Dr. H. K. V. Soltau: Scope and Effect of Institutional Treatment in Alcoholism. Discussion to be introduced by Professor A. D. Macdonald: Pharmacology of Addiction.

UNIVERSITY COLLEGE, LONDON.—At Anatomy Theatre, 1.15 p.m., Professor J. B. S. Haldane, F.R.S.: Physiology of Diving.

Wednesday, October 21

LONDON UNIVERSITY.—At Guy's Hospital Medical School, 5 p.m., Professor William Montagna (Brown University): The Architecture, Growth, and Differentiation of Skin.

POSTGRADUATE MEDICAL SCHOOL OF LONDON.—2 p.m., Professor N. H. Martin: Circulating Proteins in Health and Disease.

ROYAL COLLEGE OF SURGEONS OF ENGLAND.—5 p.m., Urology Lecture by Mr. L. N. Pyrah: Uretero-colic Anastomosis: Technique, Management, and After-care.

Thursday, October 22

ABERNETHIAN SOCIETY.—At St. Bartholomew's Hospital, London, E.C., 5.30 p.m., Opening Address by Sir Lionel Whitby: Devils, Drugs, and Doctors.

BRITISH POSTGRADUATE MEDICAL FEDERATION.—At London School of Hygiene and Tropical Medicine, 5.30 p.m., Professor F. G. Young, F.R.S.: Growth Hormone of the Anterior Pituitary Gland.

DURHAM UNIVERSITY: DEPARTMENT OF SURGERY.—At Royal Victoria Infirmary, Newcastle-upon-Tyne, 4.30 p.m., Rutherford Morison Centenary Lecture by Sir Gordon Gordon-Taylor: The Rutherford Morison Tradition.

HARVEIAN SOCIETY OF LONDON.—8.15 p.m., Buckston Browne Prize Essay by Dr. Sheila P. V. Sherlock: Jaundice.

HONYMAN GILLESPIE LECTURE.—At University New Buildings, Edinburgh, 5 p.m., Professor David Whitteridge, F.R.S.: Cardiovascular Disturbances in Paraplegia.

●INSTITUTE OF CARDIOLOGY, National Heart Hospital.—9.30 a.m., Professor M. L. Rosenheim: Oedema.

●INSTITUTE OF CHILD HEALTH AND INSTITUTE OF NEUROLOGY.—At National Hospital, Queen Square, W.C., 5.30 p.m., Sir Russell Brain: Aetiological Factors of "Acquired" Group (Virus Infections, Lipoidosis, Vascular, Degenerative Diseases).

INSTITUTE OF DERMATOLOGY.—5.30 p.m., Dr. R. W. Riddell: Structure and Classification of Pathogenic Fungi.

INSTITUTE OF LARYNGOLOGY AND OTOLOGY.—5 p.m., Professor F. C. Ormerod: Tuberculous Disease of the Upper Air Passages.

LIVERPOOL MEDICAL INSTITUTION.—8 p.m., Professor J. S. Young: Dissemination of Malignant Tumours.

ST. ANDREWS UNIVERSITY.—At Physiology Department, Dundee, 5 p.m., Dr. K. G. Lowe: Acute Renal Failure.

ST. GEORGE'S HOSPITAL MEDICAL SCHOOL.—5 p.m., Dr. J. H. Paterson: neurology demonstration.

SOCIETY FOR CULTURAL RELATIONS WITH THE U.S.S.R.—7.30 p.m., Dr. B. Kirman: Impressions of the Soviet Health Services. Report of Visit to the Soviet Union.

Friday, October 23

INSTITUTE OF DERMATOLOGY.—5.30 p.m., clinical demonstration by Dr. H. J. Wallace.

●INSTITUTE OF OBSTETRICS AND GYNAECOLOGY.—At Queen Charlotte's Hospital, 4.30 p.m., Mr. A. W. Bourne: Conservatism in Obstetrics.

LEEDS AND WEST RIDING MEDICO-CHIRURGICAL SOCIETY.—Mr. F. P. Raper: Recent Advances in the Treatment of Some Common Urological Diseases.

LONDON ASSOCIATION OF THE MEDICAL WOMEN'S FEDERATION.—At Royal Free Hospital School of Medicine, 8.30 p.m., Dr. Lindsey W. Batten: Scope and Limitations of Preventive Medicine.

●POSTGRADUATE MEDICAL SCHOOL OF LONDON.—4 p.m., Professor E. J. Wayne: Clinical Evaluation of New Drugs.

ROYAL COLLEGE OF SURGEONS OF ENGLAND.—3.45 p.m., Erasmus Wilson Demonstration by Dr. G. D. Lumb: Irradiation Changes in Malignant Disease.

ROYAL EYE HOSPITAL.—5.30 p.m., Dr. A. A. Guild: The Myopathies.

APPOINTMENTS

East Anglian Regional Hospital Board.—F. Alberts, M.B., B.S., D.A., Consultant Anaesthetist in Wisbech and Doddington Area; C. C. Smith, M.B., B.S., D.M.R.D., Assistant Radiologist in Peterborough and Stamford Area; M. Barry, M.D., M.R.C.P.I., Assistant Chest Physician at Ipswich Area Chest Clinic; D. P. F. Embleton, M.R.C.S., L.R.C.P., Senior Registrar in Chest Medicine at Ipswich Area Chest Clinic; T. J. Sullivan, M.B., B.Chir., Medical Registrar at Peterborough Memorial Hospital; S. K. Chatterjee, M.B., B.S., F.R.C.S., Surgical Registrar, Peterborough Memorial Hospital; K. N. Kamdar, M.B., B.S., D.M.R.D., Registrar in Radiology, East Suffolk and Ipswich Hospital; A. J. Abraham, F.R.C.S., Registrar in Plastic Surgery, West Norwich Hospital; J. V. Fennell, M.B., B.Ch., D.P.M., Consultant Psychiatrist in Ipswich Mental Hospital Group; J. L. Dunscombe, M.B., Ch.B., Assistant Pathologist to Hospitals in West Suffolk Area; L. Capper, M.B., B.S., M.R.C.P., Senior Registrar in Chest Medicine at Cambridge Chest Clinic; M. A. Birnstingl, M.B., B.S., F.R.C.S., Surgical Registrar, J. M. Brownlie, L.R.C.P.&S.Ed., Orthopaedic Registrar, and E. R. S. Hooper, M.B., B.S., D.A., Anaesthetic Registrar, all three at Norfolk and Norwich Hospital; D. D. Guha, M.B., Surgical Registrar at West Norfolk and King's Lynn Hospital.

South-western Regional Hospital Board.—E. Fischer, Ph.D., L.R.C.P.& S.I., Senior Hospital Medical Officer, Royal Western Counties Institution, Starcross, Devon; R. C. Allison, M.B., B.Chir., D.A., Assistant Anaesthetist (S.H.M.O.) to West Cornwall Clinical Area; J. A. Smith, M.B., B.Chir., F.F.R., D.A., Consultant Anaesthetist to Plymouth Clinical Area; F. S. W. Brimblecombe, M.D., M.R.C.P., D.C.H., Consultant Paediatrician to Exeter Clinical Area; H. P. Guerrier, M.B., B.S., F.R.C.S., Consultant Surgeon to Exeter Clinical Area (Torquay and Newton Abbot Hospitals); Kathleen J. Harrison, M.B., Ch.B., D.Obst.R.C.O.G., D.C.H., Registrar in Pathology, Frenchay Hospital, Bristol; A. G. G. Toomey, M.R.C.S., L.R.C.P., D.M.R.D., Senior Registrar in Radiology to North Gloucestershire Clinical Area; S. R. Hewitt, M.B., B.S., D.Obst.R.C.O.G., M.R.C.O.G., Senior Registrar in Obstetrics and Gynaecology, Plymouth Clinical Area; P. W. Seargeant, M.B., Ch.B., F.R.C.S., Surgical Registrar to Cossham/Frenchay Group of Hospitals, Bristol; Patricia M. Gilbert, M.B., Ch.B., Registrar to South-west Regional Blood Transfusion Centre, Southmead, Bristol; R. L. Batten, F.R.C.S., Surgical Registrar, Weston-super-Mare Hospital; R. M. Norman, M.D., M.R.C.P., D.P.M., Research Consultant in Neuropathology; A. G. Freeman, M.D., M.R.C.P., Senior Registrar in General Medicine, Exeter Clinical Area; S. Mallick, L.R.C.P.& S.Ed., D.Obst.R.C.O.G., Registrar in Obstetrics and Gynaecology, Gloucestershire Royal Hospital, Gloucester; P. A. Oye Anyaegbunam, M.B., B.Ch., D.L.O., Ear, Nose, and Throat Registrar, South Devon and East Cornwall Hospital, Plymouth.

Swann, W. G., M.D., D.P.H., D.Obst.R.C.O.G., Medical Officer of Health and Port Medical Officer of Belfast.

BIRTHS, MARRIAGES, AND DEATHS

BIRTHS

Eedy.—On September 16, 1953, to Françoise, wife of Dr. B. N. Eedy, 39, King Edward Avenue, Mansfield, Notts, a brother for Bernardine and Patrick—Michel.

Graves.—On October 3, 1953, at Kitts Croft, Writtle, Essex, to Valerie (formerly Scotland), M.B., B.S., wife of Dr. J. C. Graves, a son.

Macaskill.—On September 28, 1953, at the Boston General Hospital, to Daphne, wife of Dr. John S. M. Macaskill, 2, Church View, Coningsby, Lincs, a daughter—Elizabeth Mary.

Mills.—On October 1, 1953, in Birmingham, to Dr. Margaret J. Mills, wife of Wilfrid Mills, F.R.C.S., a son.

Ogan.—On October 4, 1953, at Queen Charlotte's Hospital, Goldhawk Road, London, W., to Oyidia, wife of O. Kesandu Ogan, M.R.C.S., L.R.C.P., D.R.C.O.G., a daughter.

Roddie.—On September 24, 1953, at Johnstone House, Royal Maternity Hospital, Belfast, to Alix P. M. Rodie (formerly Hurst), M.B., B.S., wife of T. Wilson Roddie, M.B., B.Ch., M.R.C.O.G., a daughter.

Shaw.—On October 5, 1953, to Joyce (formerly Dodds), M.B., B.S., wife of Kenneth V. Shaw, A.R.I.B.A., of Quarry End, Walworth, Darlington, Co. Durham, a son.

Shepherd.—On October 1, 1953, at Enfield, Middlesex, to Demetra, wife of Henry Robert Shepherd, D.S.C., M.B., Ch.B., a second son—Michael Charles.

Slovick.—On September 28, 1953, at King's College Hospital, London, S.E., to Pamela, wife of David Slovick, M.R.C.S., L.R.C.P., a daughter.

DEATHS

Alden.—On September 14, 1953, John Wenham Alden, M.B., B.Ch., of 186, Woodstock Road, Oxford.

Barer.—On September 22, 1953, at 29, The Avenue, Hitchin, Herts, Max Barer, M.B., B.S.

Bartlett.—On September 15, 1953, at Kisumu, Kenya, James Henry Bartlett, B.M., B.Ch., aged 44.

Clark.—On September 15, 1953, at his home, Langdon Lodge, Churston Ferrers, Devon, John Clark, M.B., C.M., formerly of Radcliffe, Lancs, aged 82.

Dow.—On September 20, 1953, in a nursing-home, Edinburgh, Robert Charles Scott Dow, L.R.C.P.&S.Ed., L.R.F.P.S., F.D.S. R.C.S.Ed., of 4, Peel Terrace, Edinburgh.

Gibson.—On September 13, 1953, at Cartref, Terrick, Aylesbury, Bucks, Michael Joseph Gibson, M.D., F.R.C.O.G., late of Dublin, aged 77.

James.—On September 18, 1953, Robert Blake James, M.R.C.S., L.R.C.P., of Riverside, Hungerford, Berks, aged 84.

Jaques.—On September 20, 1953, at Worthing Hospital, Robert Jaques, F.R.C.S., of Worthing, Sussex.

Kahan.—On September 16, 1953, Nest Kahan (formerly Thomas), M.B., B.S., D.P.M., D.C.H., formerly of Kingston, Surrey.

Keats.—On September 18, 1953, Bertram Abbott Keats, L.M.S.S.A., of 37, The Ridgeway, Finchley, London, N., aged 71.

Kennedy.—On September 15, 1953, at Boughton-under-Blean, Faversham, Kent, Arthur Kennedy, M.C., M.B., Ch.B., aged 61.

Scott.—On October 2, 1953, at his home, 22, Longton Avenue, Manchester, James Murray Scott, M.B., Ch.B., aged 44.

Scroggie.—On September 20, 1953, in a nursing-home, Stirling, William Reith John Scroggie, C.I.E., M.R.C.S., L.R.C.P., Colonel, I.M.S., retired, of Abbotsford Lodge, Callander, Perthshire.

Wills.—On September 22, 1953, Ronald Leonard Wills, M.R.C.S., L.R.C.P., of Sheerness, Kent, formerly of The Lizard, Cornwall.

Wright.—On August 22, 1953, at his home, Takapuna, New Zealand, John Edward Wright, M.B.E., F.R.C.S.Ed., F.A.C.S., L.D.S. R.C.S.

Any Questions?

Correspondents should give their names and addresses (not for publication) and include all relevant details in their questions, which should be typed. We publish here a selection of those questions and answers which seem to be of general interest.

Protection by A.T.S.

Q.—*For how long does the usual prophylactic dose of A.T.S. (say 1,500 I.U.) confer protection against tetanus arising from (a) infected wounds, and (b) further infected wounds received several days later?*

A.—(*a*) A single prophylactic dose of 1,500 I.U. (1950), which is equivalent to 3,000 I.U. (1928), is regarded by many surgeons as adequate insurance against the risk of tetanus. The protection is usually at its maximum two or three days after a subcutaneous or intramuscular injection of antitoxic serum, and then slowly diminishes as the A.T.S. is eliminated; it is probably very low after about three weeks. Occasionally, however, the duration of adequate immunity may be only a few days in some sensitive persons who have had previous injections of horse serum. If it is desired to prolong the period of protection, some surgeons double or treble the usual dose for cases considered to be tetanus-prone—for example, those with dead tissue or foreign bodies in wounds, particularly when there has been delay between wounding and efficient surgical treatment. But the more usual practice in such cases is to give two further doses, each of 1,500 I.U., at weekly intervals. There is no hard-and-fast rule. Children may be given the same doses as adults.

To-day emphasis is being laid increasingly on active immunization with tetanus toxoid. The first dose of toxoid may be injected, if desired, six weeks after the last dose of antitoxic serum.

(*b*) It is probably unwise to assume that the protection afforded by antitoxin, given more than a day or so previously, will be adequate in all persons. Another dose of serum is indicated.

Folic Acid and Diarrhoea

Q.—*It has been my experience that folic acid checks the diarrhoea in both sprue and idiopathic steatorrhoea, often before there is any recognizable improvement in the blood picture. What is the explanation of this? On the contrary, I have failed to observe any improvement in the diarrhoea of coeliac disease in children when this is treated with folic acid. Why is this?*

A.—During the war it was discovered that large doses of sulphaguanidine were effective in the diarrhoea of sprue, even though no pathogens were present. The same was true of crude liver extract. It is probable that the common action of these three agents—sulphonamides, liver, and folic acid—is a change of the intestinal flora to one which is less irritant to the mucosa. Cortisone may act in the same way or, more probably, by diminishing the irritability of the mucosa. At any rate, it is clear that, although diarrhoea, anaemia, and the defect in fat absorption are roughly parallel, they may diverge, particularly in the early stages of treatment. Diarrhoea usually improves first, next anaemia, and only after a long interval fat absorption.

It is probable that the pathology of coeliac disease is somewhat different from that of sprue, sensitivity to foods—for example, wheat gluten—being more important than disturbance in the intestinal flora. Moreover, the anaemia of coeliac disease is rarely megaloblastic. Deficiency of folic acid *per se* probably favours diarrhoea, as is suggested by the intestinal upsets of untreated pernicious anaemia. Finally, the consequential effects on the diarrhoea of improvement in the anaemia are not to be despised.

Homosexual Tendencies in Middle Age

Q.—*What treatment is advised for a man in his fifties with homosexual tendencies which have been getting worse during the last year? This is causing him considerable worry. He is married, and has two children. He is physically fit, and has no signs of prostatic disease.*

A.—Your questioner does not make it clear whether conscious homosexual tendencies were present before the patient reached his fifties, or whether they represent a new orientation. In the former case, no help could be anticipated from psychotherapy. In the latter case, it would be essential to establish the diagnosis (that is, the reason for hitherto repressed tendencies of this kind complicating the psychosexual picture) before thinking of treatment.

In either case, should moral and social considerations demand it, "biochemical castration"—that is, planned extinction of the sexual libido—should be considered. This can be effected by means of a daily dose of up to 15 mg. of stilboestrol. The possible side-effects must, however, be remembered.

Unilateral Premenstrual Mastalgia

Q.—*I have a patient who for the past year has had mastitis in the left breast coming on fourteen days after a period. It lasts until the next period starts and has gradually been getting worse, the left arm being also affected by pain. On the last occasion, the pain started again on the second day of the period (which lasts only two to three days) instead of on the fourteenth day. Latterly, she has been having some pain in the right breast too. The pain is becoming very severe and, being more or less continuous, is making her ill and depressed. She had a similar occurrence some five years ago, but this lasted some six to seven months only. Is there any treatment?*

A.—This condition is probably an exaggeration of the usual premenstrual mastalgia—an exaggeration which is part of a menstrual tension state. The breasts are often unequal in development and function, so it is common for this type of discomfort to be worse on one side. In this condition there is nearly always a background of overanxiety and frequently a fear of cancer. Indeed, the general illness and depression in this case are more likely to be the cause than the result of the pain.

The patient should be reassured and instructed to wear a well-fitting brassière. Her intake of fluid should be restricted to not more than 30 oz. (850 ml.) in 24 hours for two weeks premenstrually, and at the same time salt should be avoided as far as possible. Sodium retention is also discouraged by giving ammonium chloride 10 gr. (0.65 g.), in capsules, three times daily.

Hormone therapy is not very satisfactory for premenstrual mastalgia. Oestrogens may reduce the discomfort but at the expense of upsetting the menstrual cycle. Small doses of androgens are preferable but involve some risk of producing masculinizing effects. If the simple measures mentioned above are insufficient, then the first hormone to be tried should be ethisterone 5–10 mg. sublingually three times daily for ten days premenstrually. It is unlikely to have any undesirable side-effects and sometimes appears helpful.

Otitis Externa in the Tropics

Q.—*Why is otitis externa so common in the Tropics? How is it acquired, and what measures of prevention are recommended?*

A.—The external auditory meatus is lined with skin, and this is the only area of skin in the body which is not readily accessible for cleansing purposes. In consequence any excess of moisture either from perspiration or from water getting into the ear may cause maceration of the superficial epithelium, thus allowing organisms to gain entry and produce an acute inflammation. The common pathogens are the streptococcus, *Pseudomonas pyocyanea*, and certain fungi of the aspergillus group. Though the infecting agent is different the mechanism of production of external otitis is similar to that responsible for athlete's foot. In both the infection is in an area of skin not always readily accessible and is most commonly found in visitors to hot countries. Unlike athlete's foot, however, otitis externa does not commonly spread from one person to another.

In the very acute stage great pain and considerable constitutional disturbance may be experienced, and rest in bed with sedatives and an antibiotic may be needed. Locally a wick of wool soaked in half-strength *meta*cresyl acetate ("cresatin") and left in for a few hours will often give relief if combined with local heat. For a fungus infection 2% thymol in spirit may be needed.

The prevention of otitis externa in the Tropics is best achieved by preventing water from getting into the ear canal, and by using a mild oily mercurial solution as ear drops twice weekly (dilute mercuric nitrate ointment, 1 drachm (4 g.); expressed almond oil to 1 oz. (28.4 ml.)).

Resorcin Stains

Q.—*Please can you tell me how to remove the brown stains of resorcin from a wash-basin?*

A.—Fresh stains of resorcin may often be removed by sponging with alcohol, ether, or glycerin. Older stains should be treated with a warm 3% solution of citric acid. If the stain has seeped through cracks in the glazed surface of the porcelain, prolonged soaking may be necessary.

NOTES AND COMMENTS

Filatov's Tissue Therapy.—Dr. W. M. Corbet (of *Excerpta Medica*, Amsterdam) writes: Your expert dismisses the query about Filatov's tissue therapy ("Any Questions?" September 12, p. 634) somewhat lightly, and this misleads the questioner. Many enthusiastic reports have appeared, and continue to appear, more especially in the Italian and Spanish literature.

Our Expert writes: I am quite certain that I have not misled the questioner as to the therapeutic value of Filatov's treatment, though I may have been slightly misleading in suggesting that nearly all the references were confined to French and Russian journals. I do not think it is a matter of any importance.

Car Sickness in Small Children.—Dr. Francis Leslie (London, S.W.1) writes: Some children are completely cured of car sickness and the feeling of nausea ("Any Questions?" August 29, p. 519) if the car is earthed by means of a metal chain attached to a bumper, provided that one or two links of that chain touch the ground at slow speeds or when the car is stationary. A car, just because it is insulated by rubber tyres, must presumably possess quite an appreciable electrical charge similar to the rubber-tyred operation theatre trolley, and the fact that some children are cured of sickness and nausea by the earthing of the vehicle suggests that an electrical charge is responsible for their symptoms. I know of one firm of car agents who did not like the appearance of a chain hanging from a car delivered to one of their clients, and so they installed a metal tube under the car through which passed the earthing chain, and reserve chain could be released as the end links were worn away by contact with the road surface. Surely these facts invite fuller investigation, and I would welcome the comments of those with children who suffer from car sickness or nausea.

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